ENT SCHOLAR

VOLUME 2 ISSUE 1 2013

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PUBLISHER: DR BALASUBRAMANIAN THIAGARAJAN

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Publisher:

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Advanced anatomy of lateral nasal wall

For the endoscopic sinus surgeon

September 19, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Anatomy of lateral nasal wall

Introduction:

Anatomy of the lateral nasal wall is highly complex and variable. With the popularity of endoscopic sinus surgery a through knowledge of this complex anatomy is very vital. Highly variable anatomy and paucity of standard landmark makes this region vulnerable for complications during endoscopic sinus surgery. The learning curve for endoscopic sinus surgery is made rather steep by this highly variable anatomy [1]. Study of anatomy of lateral nasal wall dates back to Galen (AD 130-201). He described the porosity of bones in the head. Davinci in his classical anatomical drawings has illustrated maxillary sinus antrum. He also described maxillary sinus as cavities in the bone that supports the cheek [2]. Highmore (1651) described maxillary sinus anatomy. Hence it is also known as antrum of Highmore [3]. It was during the 19th century that Zuckerkandl came out with the first detailed description of maxillary sinus and its surrounding anatomy. Paranasal sinuses are four air filled cavities situated at the entrance of the upper airway. Each of these sinuses are named after the skull bone in which it is located [4].

Nasal turbinates:

The turbinates are the most prominent feature of the lateral nasal wall [5]. They are usually three or sometimes four in number. These turbinates appear as scrolls of bone, delicate, covered by ciliated columnar epithelium. These turbinates sometimes may contain an air cell, in which case it is termed as a concha.

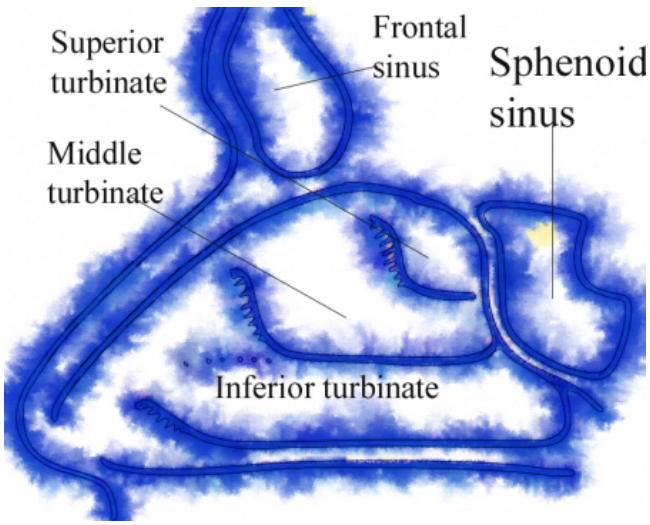
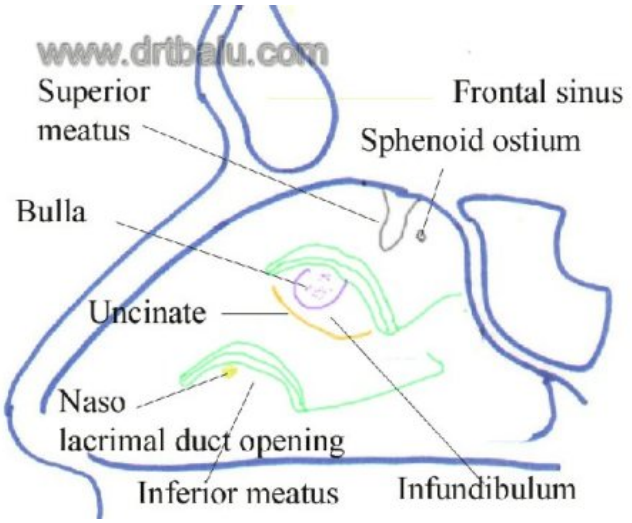


Fig. 1: Figure showing turbinates in the lateral nasal wall

These turbinates project from the lateral wall of the nose. Out of these turbinates the following are present in all individuals:

The superior, middle and inferior turbinates. A small supreme turbinate may be present in some individuals. Among these turbinates the superior and the middle turbinates are components of the ethmodial complex where as the inferior turbinate is a separate bone. Commonly a prominence may be seen at the attachment of the middle turbinate.



lateral wall of nose after removal of turbinates

This prominence is known as the agger nasi cell. This prominence varies in size in different individuals. These agger nasi cells overlie the lacrimal sac, separated from it just by a thin layer of bone. Infact this agger nasi cell is considered to be a remnant of naso turbinal bones seen in animals. When the anterior attachment of the inferior and middle turbinates are removed, the lacrimal drainage system and sinus drainage system can be clearly seen.

The inferior turbinate is a separate bone developed embryologically from the maxilloturbinal bone.

The inferior meatus is present between the inferior turbianate and the lateral nasal wall. The nasal opening of the naso lacrimal duct opens in the anterior third of the inferior meatus. This opening is covered by a mucosal valve known as the Hassner's valve. The course of the naso lacrimal duct from the lacrimal sac lie under the agger nasi cell.

The middle meatus lie between the middle turbinate and the lateral nasal wall. The middle turbinate is part of the ethmoidal complex. The sinuses have been divided into the anterior and posterior groups. The anterior group of sinuses are frontal, maxillary and anterior ethmoidal sinuses. These sinuses drain into the middle meatus, i.e. under the middle turbinate. The middle meatus hosts from anterior to posterior the following structures:

1. Agger nasi
2. Uncinate process
3. Hiatus semilunaris
4. Ethmoidal bulla
5. Sinus lateralis
6. Posterior fontanelle

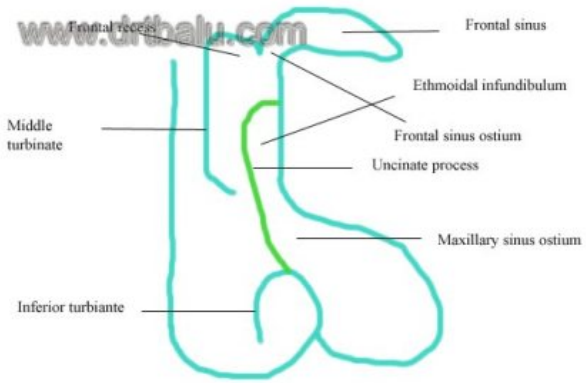
Uncinate process: actually forms the first layer or lamella of the middle meatus. This is the most stable landmark in the lateral nasal wall. It is a wing or boomerang shaped piece of bone. It attaches

anteriorly to the posterior edge of the lacrimal bone, and inferiorly to the superior edge of the inferior turbinate [6]. Superior attachment of the uncinate process is highly variable, may be attached to the lamina palyracea, or the roof of the ethmoid sinus, or sometimes to the middle turbinate. The configuration of the ethmoidal infundibulum and its relationship to the frontal recess depends largely on the behavior of the uncinate process. The Uncinate process can be classified into 3 types depending on its superior attachment.

The anterior insertion of the uncinate process cannot be identified clearly because it is covered with mucosa which is continuous with that of the lateral nasal wall. Sometimes a small groove is visible over the area where the uncinate attaches itself to the lateral nasal wall. The anterior convex part forms the anterior boundary of the ostiomeatal complex. It is here the maxillary, anterior ethmoidal and frontal sinuses drain. Uncinate process can be displaced medially by the presence of polypoidal tissue, or laterally against the orbit in

individuals with maxillary sinus hypoplasia [7]. Removing of this piece of bone is the most important step in Endoscopic sinus surgery.

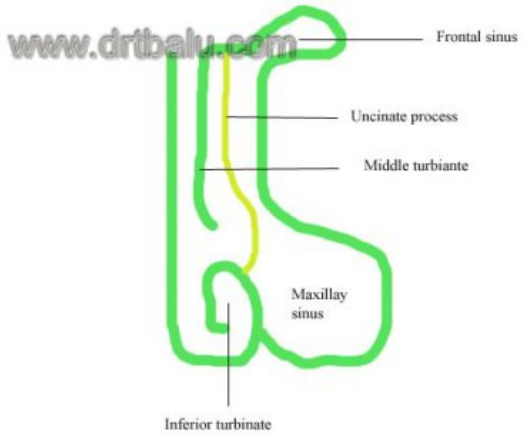
Type I uncinate: Here the uncinate process bends laterally in its upper most portion and inserts into the lamina papyracea. Here the ethmoidal infundibulum is closed superiorly by a blind pouch called the recessus terminalis (terminal recess). In this case the ethmoidal infundibulum and the frontal recess are separated from each other so that the frontal recess opens into the middle meatus medial to the ethmoidal infundibulum, between the uncinate process and the middle turbinate. The route of drainage and ventilation of the frontal sinus run medial to the ethmoidal infundibulum.



Type I uncinate insertion

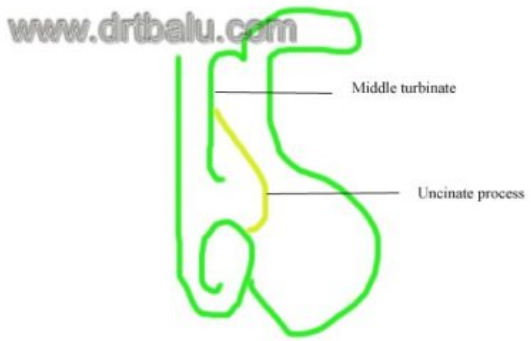
Type II uncinate: Here the uncinate process extends superiorly to the roof of the ethmoid. The frontal sinus opens directly into the ethmoidal infundibulum. In these cases a disease in the frontal recess may spread to involve the ethmoidal infundibulum and the maxillary sinus secondarily. Sometimes the superior end of the uncinate process may get divided into three branches one getting attached to the roof of the ethmoid, one getting attached to the lamina papyracea, and the last getting attached to the middle turbinate.

Type II uncinate insertion

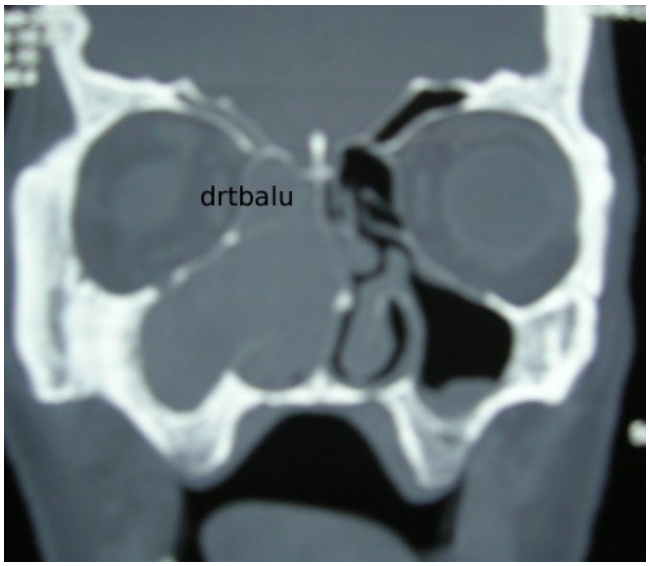


Type III uncinate process: Here the superior end of the uncinate process turns medially to get attached to the middle turbinate. Here also the frontal sinus drains directly into the ethmoidal infundibulum.

Rarely the uncinate process itself may be heavily pneumatised causing obstruction to the infundibulum.



Type III uncinate insertion



Polyp seen pushing the uncinate medially

Hypoplasia of maxillary sinus seen pushing the uncinate laterally

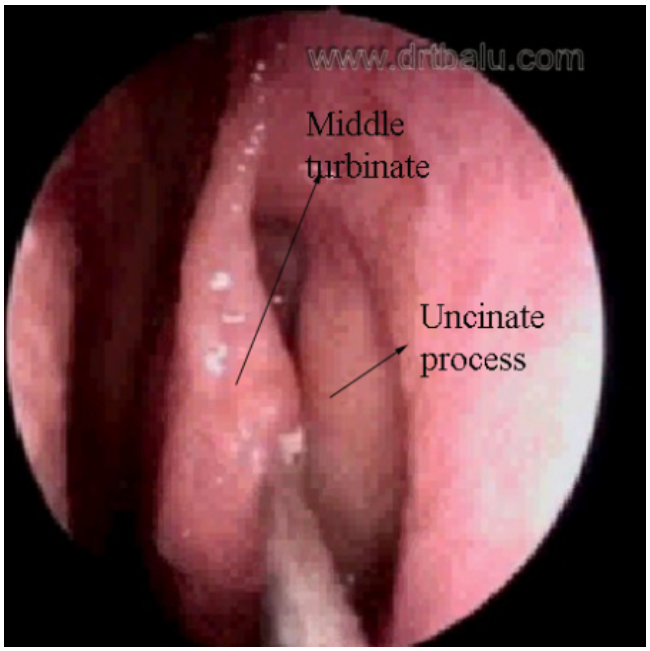


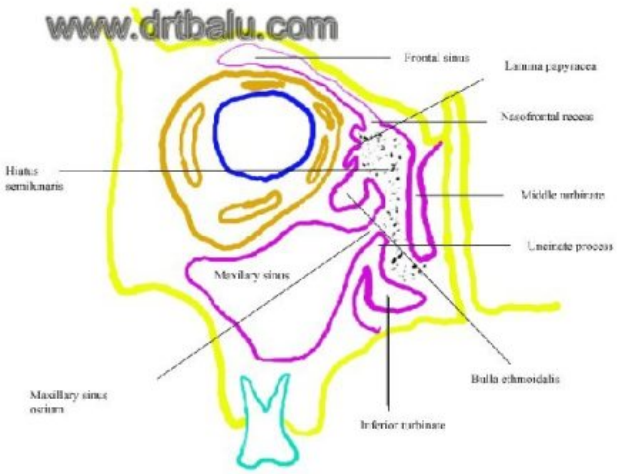
Image showing uncinate process

Removal of uncinate process reveals the natural ostium of the maxillary sinus. This is another vital landmark in the lateral nasal cavity. The superior wall of the natural ostium of the maxillary sinus is at the level of floor of the orbit.Agger nasi: This is a latin word for “Mound”. This area refers to the most superior remnant of the first ethmoturbinal which presents as a mound anterior and superior to the insertion of middle turbinate.Depending on the pneumatization of this area may reach up to the level of lacrimal fossa thereby causing narrowing of frontal sinus outflow tract. Ethmoidal infundibulum: is a cleft like space, which is three dimensional in the lateral wall of the nose. This structure belongs to the anterior ethmoid. This space is bounded medially by the uncinate process and the mucosa covering it. Major portion of its lateral wall is bounded by the lamina papyracea, and the frontal process of maxilla to a lesser extent. Defects in the medial wall of the infundibulum is covered with dense connective tissue and periosteum. These defects are known as anterior and poterior fontanelles. Anteriorly the ethmoidal infundibulum ends blindly in an acute angle.

Figure showing large agger nasi air cell

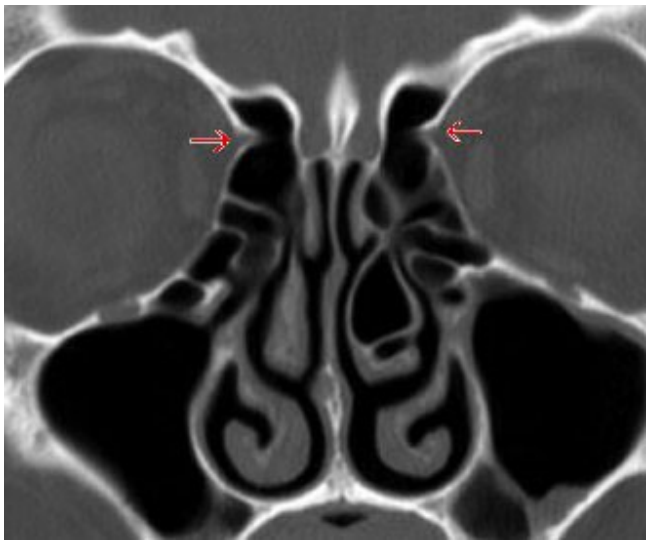
Bulla ethmoidalis: This is derived from Latin. Bulla means a hollow thin walled bony prominence. This is another landmark since it is the largest and non variant of the aircells belonging to the anterior

ethmoidal complex. This aircell is formed by pneumatization of bulla lamella (second ethmoid basal lamella). This air cell appears like a bleb situated in the lamina papyracea. Some authors consider this to be a middle ethmoid cell. If bulla extends up to the roof of ethmoid it can form the posterior wall of frontal recess. If it does not reach up to the level of skull base then a recess can be formed between the bulla and skull base. This recess is known as suprabullar recess. If the posterior wall of bulla is not in contact with basal lamella then a recess is formed between bulla and basal lamella. This recess is known as retrobullar recess / sinus lateralis. This retrobullar recess may communicate with the suprabullar recess. Osteomeatal complex: This term is used by the surgeon to indicate the area bounded by the middle turbinate medially, the lamina papyracea laterally, and the basal lamella superiorly and posteriorly. The inferior and anterior borders of the osteomeatal complex are open. The contents of this space are the aggernasi, nasofrontal recess (frontal recess), infundibulum, bulla ethmoidalis and the anterior group of ethmoidal air cells.This is infact a narrow anatomical region consisting of : 1. Multiple bony structures (Middle turbinate, uncinate process, Bulla ethmoidalis) 2. Air spaces (Frontal recess, ethmoidal infundibulum, middle meatus) 3. Ostia of anterior ethmoidal, maxillary and frontal sinuses. In this area, the mucosal surfaces are very close, sometimes even in contact causing secretions to accumulate. The cilia by their sweeping movements pushes the nasal secretions. If the mucosa lining this area becomes inflamed and swollen the mucociliary clearance is inhibited, eventually blocking the sinuses. Some authors divide this osteomeatal complex into anterior and posterior. The classic osteomeatal complex described already has been described as the anterior osteomeatal complex, while the space behind the basal lamella containing the posterior ethmoidal cells is referred to as the posterior ethmoidal complex, thus recognising the importance of basal lamella as an anatomical landmark to the posterior ethmoidal system. Hence the anterior and the posterior osteomeatal complex has separate drainage systems. So when the disease is limited to the anterior compartment of the osteomeatal complex, the ethmoid cells can be opened and diseased tissue removed as far as the basal lamella, leaving the basal lamella undisturbed minimising the risk during surgery.Hiatus semilunaris: Lies between the anterior wall of the Bulla and the free posterior margin of the uncinate process. This is infact a two dimensional space. Through this hiatus a cleft like space can be entered. This is known as the ehtmoidal infundibulum. This ethmoidal infundibulum is bounded medially along its entire length by the uncinate process and its lining mucosa. The lateral wall is formed by the lamina papyracea of the orbit, with participation from the frontal process of the maxilla and the lacrimal bone. The anterior group of sinuses drain into this area. Infact this area acts as a cess pool for all the secretions from the anterior group of sinuses.



Osteomeatal complex

Concha bullosa: Sometimes middle turbinate may become pneumatized. This pneumatization is known as concha bullosa. This process of pneumatization starts either from frontal recess or agger nasi air cells. This is usually considered to be a normal variant. Sometimes this pneumatization may become so extensive that it could cause obstruction in osteomeatal complex [8].



Coronal CT showing concha bullosa

References

1. Ohnishi T, Tachibana T, Kaneko Y, et al. High-risk areas in endoscopic sinus surgery and prevention of complications. Laryngoscope. 1993;103: 181–5

2. Stoney P, MacKay A, Hawke M.The antrum of Highmore or of da Vinci? J Otolaryngol. 1991 Dec ;20(6):456-8

3. Blanton PL, Biggs NL (1969) Eighteen hundred years of controversy: the paranasal sinuses. Am J Anat 124(2):135–47.

4. Graney DO, Rice DH, Paranasal sinuses anatomy. In: Cummings CW, Fredrickson JM, Harker LA et al (1998)Otolaryngology Head and Neck Surgery. Mosby, 3rd edn.

5. Bodino C, Jankowski R, Grignon B et al (2004) Surgical anatomy of the turbinal wall of the ethmoidal labyrinth.Rhinology 42(2):73–80.

6. Bolger WE, Anatomy of the Paranasal Sinuses. In: Kennedy DW, Bolger WE, Zinreich J (2001) Diseases of the sinuses, Diagnosis and management. B.C. Decker

7. http://jorl.net/index.php/jorl/article/view/47

8. Joe JK, Ho SY, Yanagisawa E (2000) Documentation of variations in sinonasal anatomy by intraoperative nasal endoscopy. Laryngoscope 110(2):229–35.

Allergic rhinitis an overview

September 3, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Abstract:

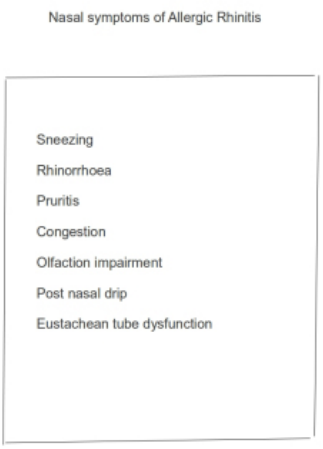
Rhinitis is classically defined as inflammation of nasal mucosa characterised by a symptom complex which includes sneezing, nasal congestion, itching and rhinorrhoea. Allergy happens to be the most common cause for rhinitis affecting approximately 20% of the population. Eventhough allergic rhinitis is not a life threatening disorder it causes a fair degree of morbidity and reduction in the quality of life. This also happens to be one of the common causes of rhinosinusitis.

Introduction:

Rhinitis is defined as inflammation of nasal mucous membrane characterised by symptom complex which includes sneezing, nasal congestion, itiching and rhinorrhoea [1]. This condition is rather common affecting nearly 20% of population. Eventhough this is not a life theatening disorder it causes a tremendous impact on the quality of life[2]. The classic feature of allergic rhinitis is IgE mediated Type I hypersensitivity reaction. Even ingested food can cause allergic reaction in the nasal mucous membrane due to the similarites with inhales allergens. The symptom complex which are features of allergic rhinitis can be classified into nasal and non nasal symptoms[3].

Immunological mechanisms responsible for allergic rhinitis:

Nasal symptoms



Allergic sensitization of nasal mucosa occurs in a series of well caliberated steps [4].

Non Nasal symptoms

Lacrimation

Conjunctivitis

Itchy eyes

Fatigue

Sleep disturbances

Depression

Headache

Palatal pruritus

Ear fullness

Otalgia

Midface pressure

Step I: This step is also known as antigen processing stage. This is characterised by antigen processing by antigen processing cells / macrophages. Antigen on entering the nasal cavity is ingested by these cells (dendritic antigen processing cells / macrophages). The ingested antigen undergoes a series of processing stages within the cell.

Step II: Exposure of HLA Class II receptors on the antigen processing cell. Successful antigen processing within the antigen processing cell / macrophages leads to exposure of HLA class II receptors on their cell surfaces.

Step III: In this step the activated antigen presenting cell comes into contact with Helper T cells via HLA Class II receptors present on their surface. These Helper T cells belong to $CD4^+$ T_H2 T- Helper cell category. This interaction activates the T_H2 Helper T cells.

Step IV: This step is characterised by release of cytokines like IL-4 and IL-13 which stimulates more Helper T cells to respond to the antigen load by recruiting them. These cytokines also causes B lymphocytes to differentiate into Plasma cells. These plasma cells secrete Ig E antibodies specific to the antigen.

Step V: Attachment of IgE antibodies to mast cells. This stage causes release of histamine from the activated mast cells by antigen specific IgE antibodies. Mast cells are known to contain preformed immune mediators like histamine, kinins and proteases.

These 5 stages conclude the early phase of allergic reaction. This response occurs within about 10-30 minutes following allergen exposure. The degranulating mast cells present in the nasal mucosa increases membrane permeability causing mucosal oedema, irritation, nasal pruritus, sneezing, and rhinorrhoea.

Late phase of allergic reaction:

This phase occurs between 4-8 hours following antigen exposure. This phase is cause due to:

Chemotaxis

Migration of neutrophils, basophils, eosinophils and macrophages to the nasal submucosa

Cytokines and chemokines released from these cells not only maintains the inflammatory state of nasal mucosa but also causes irrepairable damage to it.

Type of allergic rhinitis:

Allergic rhinitis has been divide into two types:

Seasonal allergic rhinitis

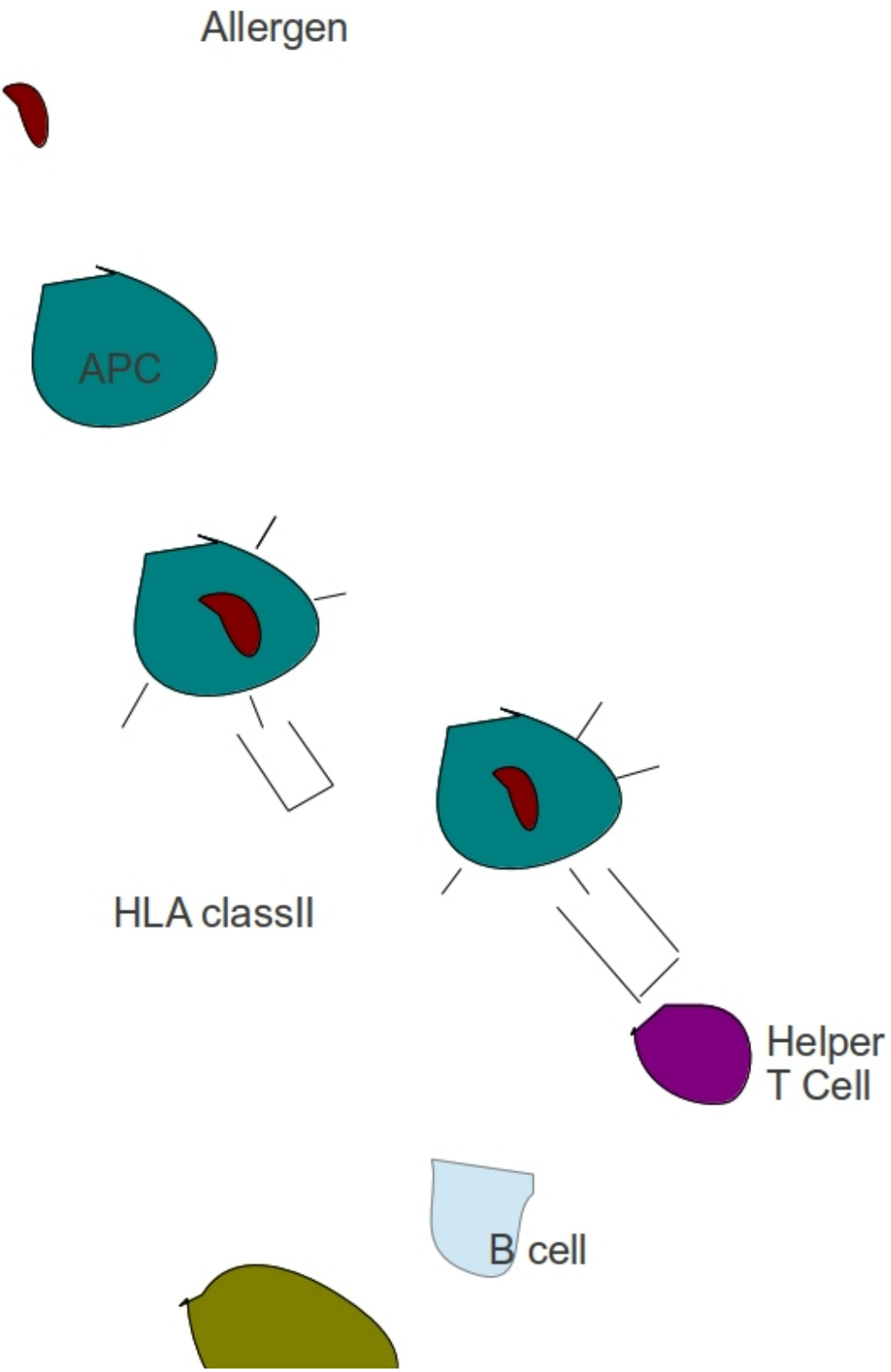
Perennial allergic rhinitis

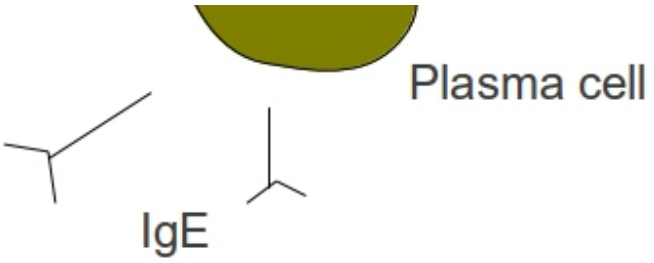
Seasonal allergic rhinitis:

This condition is also known as Hay fever. This condition occurs during different seasons depending upon the inciting allergen. Typical allergens causing Hay fever according to seasons include: tree pollen during spring, grass pollen in summer and weed pollen during fall. History is virtually diagnostic.

Perennial allergic rhinitis:

This is a more chronic form of allergic rhinitis. Symptoms are present in varying degrees right through the year. Offending allergin include moulds, cockroaches, mites and animal dander.





Clinical features:

Most of these patients develop symptoms by the age of 20[5].

Family history of allergic rhinitis is very important. Allergic rhinitis has positive genetic component[6].

Symptoms include:

Nasal block

Rhinorrhoea

Watery eyes

Itchy eyes

Repeated episodes of sneezing

Intense nasal itching will cause the patient to keep stroking the dorsum of nose. This is known as “Allergic Salute”. Continuous stroking of the dorsum of the nose will cause a horizontal skin crease to develop. This is known as Darrier’s line.

On clinical examination nasal mucosa may appear as pale and boggy. Some of these patients may even develop nasal polyposis.

Presence of allergic shiners: This is caused due to persistent nasal congestion leading on to lower eyelid oedema.

Development of creases over lower eyelid due to persistent Muller muscle spasm. This is known as Dennie’s line.

Posterior pharyngeal wall mucosa will show cobblestoning. Some of these patients may also have prominent lateral pharyngeal bands.

Persistence of nasal allergy will lead to prolonged mucociliary movement abnormality causing seconday rhinosinusitis.

Management:

Allergic rhinitis is best managed medically. Antihistamines and nasal decongestants may help during acute phases of the disorder. Topical corticosteroid spray will help in prolonged remission.

Leukotreine receptor antogonist like Montelukast[7]inhibits cysteinyl leukotriene receptor there by preventing the effects of leukotriene releaed by mast cell degranulation process.

References

1. Togias AG. Systemic immunologic and inflammatory aspects of allergic rhinitis. J Allergy Clin Immunol. Nov 2000;106(5 Suppl):S247-50

2. Summary of health statistics for U.S. adults: national health interview survey, 2002. Available at: http://www.cdc.gov/nchs/fastats/allergies.htm. Accessed August 1, 2007.

3. Druce HM. Allergic and nonallergic rhinitis. In: Middleton EM Jr, Reed CE, Ellis EF, Adkinson NF Jr, Yunginger JW, Busse WW, eds. Allergy: Principles and Practice. 5Th ed. St. Louis, Mo: Mosby Year-Book; 1998:1005-16.

4. Skoner DP. Allergic rhinitis: definition, epidemiology, pathophysiology, detection, and diagnosis. J Allergy Clin Immunol. Jul 2001;108(1 Suppl):S2-8

5. Dykewicz MS, Fineman S, Skoner DP, Nicklas R, Lee R, Blessing-Moore J. Diagnosis and management of rhinitis: complete guidelines of the Joint Task Force on Practice Parameters in Allergy, Asthma and Immunology. American Academy of Allergy, Asthma, and Immunology. Ann Allergy Asthma Immunol. Nov 1998;81(5 Pt 2):478-518.

6. Torres-Borrego J, Molina-Teran AB, Montes-Mendoza C. Prevalence and associated factors of allergic rhinitis and atopic dermatitis in children. Allergol Immunopathol (Madr). Mar-Apr 2008;36(2):90-100.

7. Nayak A, Langdon RB. Montelukast in the treatment of allergic rhinitis: an evidence-based review. Drugs. 2007;67(6):887-901

Anatomy of Paranasal sinuses

November 8, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Abstract

Paranasal sinuses are air filled hollow sacs seen around the skull bone. These sacs precisely surround the nasal cavity. There are four paired sinuses surrounding the nasal cavity. This article attempts to trace the history of anatomy of paranasal sinuses from early 16th century till date. The advent of nasal endoscopes have added another dimension to the anatomical study of paranasal sinuses. The entire subject of anatomy of paranasal sinuses has been rewritten after endoscopes were started to be used commonly.

Introduction:

Precise understanding of anatomy of paranasal sinuses is an important prerequisite in avoiding complications during endoscopic sinus surgery. Common complications following endoscopic sinus surgery are caused by inadequate understanding of the highly complex and variable anatomy of paranasal sinuses.

History:

In ancient times nasal sinuses were considered to be a system of hollow spaces through which secretions from brain drained[1]. Leonardo Davinci in 1489 was the first to prepare detailed anatomical drawings of paranasal sinuses which is still considered to be accurate till date[2]. This drawing became accessible to the scientific community only as late as 1901.

Davinci described maxillary sinus as the cavity within the bone that supports the cheek. This is still an undisputed fact. This description comes from a versatile artist cum scientist. Davicini was actually an unique combination of an artist cum scientist. His scientific descriptions were backed by his ability to draw astounding images.

Fig. 1: Portrait of Davinci

Literature on the anatomy of paranasal sinuses dates back to 130 AD. Galen during this period documented the presence of porosities around skull bones. Highmore in 1651 gave an accurate description of maxillary sinus. Maxillary sinus is still known as antrum of Highmore in honour of his description[3]. It was during early 19th century Zukerkandl came out with detailed anatomical

description of paranasal sinuses. He was also able to demonstrate the various pathologies affecting these air filled sacs. It was C. V. Schneider of Germany who suggested that mucous was not a product of brain but on the contrary was secreted by the lining mucosa of paranasal sinuses. In his honor the nasal mucous membrane is known as schneiderian membrane.

The advent of CT scan has thrown a lot of light as far as paranasal sinus anatomy is concerned. It should also be pointed out even after the advent of advanced imaging techniques we have not added much to the descriptions already made by Onodi, Grunwald and Zuckerkandl . This is an apt testimony to the ability of our forefathers[4].

Definition:

Paranasal sinuses are air filled sacs found in the skull bone. These sacs infact surround the nasal cavity. There are 4 paired sinuses. They are:

1. Maxillary sinuses

2. Frontal sinuses

3. Ethmoidal sinuses

4. Sphenoidal sinuses

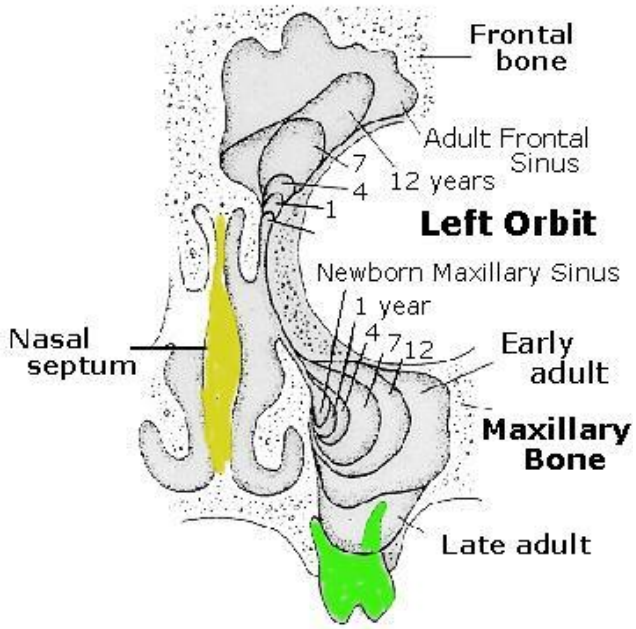
Each of these sinuses are named according to the facial bones

Maxillary sinus (Antrum of Highmore): These paired sinuses lie under the cheek. It is the largest of the group of para nasal sinuses. The capacity of the maxillary sinus is roughly 1 fluid ounze (30ml). It is more or less shaped like a pyramid. This is the first sinus to develop

Base (medial wall): The base of the pyramid corresponds to the lateral nasal wall. This wall has its convexity facing the sinus. The central portion of the base is very thin, and in some areas could even be membranous. The natural ostium of this sinus is present in this wall. It is present more towards the roof of the sinus cavity than its base.

Anterior: Wall corresponds to the facial surface of the superior maxilla. Over the canine fossa it is only 2mm in thickness. It is though this canine fossa area that maxillay antrum is entered during Caldwell Luc surgery.

Fig. 2: Development of maxillary sinus



Boundaries of Canine fossa:

Inferior: Bounded by the alveolar ridge

Laterally: Bounded by the canine eminence which is caused by the canine tooth.

Superior: Infra orbital foramen

Medial: Pyriform aperture. This does not contain bone, but is lined by middle meatus mucosa, a layer of connective tissue and the sinus mucosa[6].

Posterior wall: of maxillary sinus is also known as temporal surface. It is very thick and is formed by the body of the superior portion of the maxilla.

Roof: of the sinus is formed by its thin orbital wall which is traversed by the infra orbital foramen containing the infra orbital vessels and nerves. This wall is very fragile and any disease process involving the maxilla is likely to affect the orbit through this wall. This wall is further thinned out where the infra orbital canal is present.

Floor: is formed alveolar process of the maxilla and the hard palate. The roots of the first and second molar reach up to the floor of the maxillary sinus. In children the floor lies at the same level as that of the nasal cavity. In adults it lies 5 – 10 mm below the nasal cavity. It is just separated from the floor of the sinus by a thin lamella of bone. This lamella may be dehiscent commonly. Dental infections involving the 1st and 2nd molars may involve the maxillary sinus through this thin lamella of bone.

The most common anatomical variation is the presence of infraorbital cell (Haller cell). These are pneumatized anterior ethmoidal cells projecting along the floor of the orbit. These cells when infected can compromise drainage of maxillary sinus. If this cell is present removing it during surgery will allow precise identification of floor of the orbit and the posterior wall of maxillary sinus. This is rather useful when anatomy is rather distorted due to disease.

Fig. 3: CT scan showing Haller cell

Paranasal sinuses develop from ridges and furrows in the lateral nasal wall. This development begins as early as 8th week of intrauterine life and proceeds well into early adulthood[5].

The maxillay sinus has biphasic growth. The first phase of growth occur during the first 3 years of life while the second phase occur between 7 – 18 years.

Ethmoid sinus:

The ethmoid sinus is referred to as ethmoidal labyrinth because of the complexity of anatomy, its honey combed appearance and presence of intricate pathways and blind alleys. It is situated in the anterior skull base. It is located lateral to the olfactory cleft and olfactory fossa. It is made up of complex bony labyrinth of thin walled cells. A few ethmoid cells may be present at birth. This number could easily go beyond 15 in adults. The common infections affecting the pediatric age group occur in this sinuses. In adults 6 – 10 ethmoid cells may be present. It is the most compartmentalized of all paranasal sinuses. The width of ethmoid increases from anterior to posterior because of the cone like structure of orbit.

Boundaries:

Lateral wall: is formed by the orbital plate of the ethmoid otherwise known as the lamina papyracea. This is a thin lamina of bone separating the orbit from the ethmoidal air cells. This wall could be dehiscent (normal variant). Infections involving the ethmoid air cells may spread to the orbit through this wall.

Roof: is formed by the frontal bone anteriorly, this area is known as fovea ethmoidalis[7], and by the face of sphenoid and orbital process of palatine bone posteriorly. The frontal bone component of the ethmoidal roof is very thick. The transition of this thick fovea to the thin portion of roof of ethmoid medially is very weak. This is infact the weakest portion of this area and is prone to

injuries during surgery leading on to csf leak. The anterior and posterior ethmoidal arteries run along the roof of the ethmoid from lateral to medial. These arteries are branches of opthalmic artery.

The ethmoidal cells increase in sizefrom above downwards, and from before backwards.

The cells of the ethmoid sinus are divided into two groups, the anterior and posterior group. The anterior ethmoidal cells drain into the infundibulum of the middle meatus while the posterior ethmoid cells drain into the superior meatus. The anatomy of the ethmoidal cells are highly variable, sometimes the middle turbinate may contain an air cell known as the concha bullosa. An enlarged concha bullosa may impede drainage from the middle meatus. Another common anatomical variation is the presence of agger nasi air cell. This is a large ethmoidal air cell present just anterior to the antero superior attachment of the middle turbinate. Since these cells lie in close proximity to the frontal recess area, they could impede ventilation and drainage of the frontal sinus.These agger nasi cells are commonly involved in the pathogenesis of the formation of frontal mucocele.

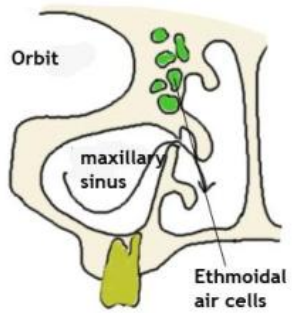


Fig. 4: Diagram showing maxillary and ethmoid sinuses

Obstruction to the frontal sinus drainage by the presence of a lage agger nasi cell may cause secretions within the frontal sinus to be dammed inside. Accumulation of mucoid secretions cause enlargement of frontal sinus. At first the frontal sinus enlarges in size by expansion of its bony walls. At a later stage bone erosion can also occur. commonly the posterior table of the frontal sinus is eroded. The anterior table also can be eroded in rare cases.

Haller cells are ethmoidal air cells belonging to the anterior ethmoidal group. These cells are also known as the infra orbital cells. Enlargement of these cells can impede the maxillary sinus drainage. Another variation is the extension of the posterior group of ethmoidal air cells supero lateral to the sphenoid sinus. These cells are known as onodi cells. These cells lieperilouslyclose to the optic nerve making them at risk during fess surgeries.

Simplified anatomy of ethmoidal sinus:

The entire anatomy of ethmoid sinus can be simplified if it is considered to be 5 obliquely oriented parallel lamellae. These lamellae are embryologically known to be derived from the ridges in the lateral nasal wall of the foetus known as ethmoturbinals. These lamellae are relatively constant and are easy to identify during surgery.

First Lamella:

This is the anterior most lamella. Anatoimically this corresponds to the uncinate process. Embryologically this represents the basal lamella of the first ethmoturbinal. This lamella is encountered by the surgeon first. It should be removed before other portions of the ethmoid sinus and its drainage system becomes visible.

Second Lamella:

This corresponds to Bulla ethmoidalis. This is the largest and most constant of the anterior ethmoidal air cells[8]. This cell was identified by Zukerkandl. This is usually round in shape with thin walls. It extends from the lamina papyraea laterally and bulges into middle meatus medially. Rarely this air cell is not pneumatized resulting in a bony projection arising from lamina papyracea. This projection is known as torus lateralis.

Third Lamella:

This is the most important of all lamella. This happens to be the ground lamella / Basal Lamella of middle turbinate. This lamella separates anterior ethmoidal air cells from posterior ethmoidal ones. This division is mandatory for the simple reason that anterior ethmoid air cells drain via the middle meatus and posterior ethmoidal air cells drain via sphenoethmoidal recess. From surgeon's point of view it is the limit for anterior ethmoidectomy. This lamella stabilizes the middle turbinate.

Fourth Lamella:

This happens to be the basal lamella of superior turbinate.

Fifth Lamella:

This is the basal lamella of supreme turbinate.

Anterior ethmoidal air cells:

Agger Nasi:

This is the anterior most of all ethmoidal air cells. The term Agger in Latin means Mound/Eminence. Endoscopically this cell is visualised as a prominence anterior to the insertion of middle turbinate. Rarely this pneumatization can involve the antero superior portion of unicinate process. Embryologically this cell is a pneumatization of superior remnant of first ethmoturbinal whorl present in the lateral wall of nasal cavity. Identification of agger nasi holds the key to access to frontal recess area. Pneumatization of agger nasi can have an impact on uncinate process insertion and patency of frontal recess area.

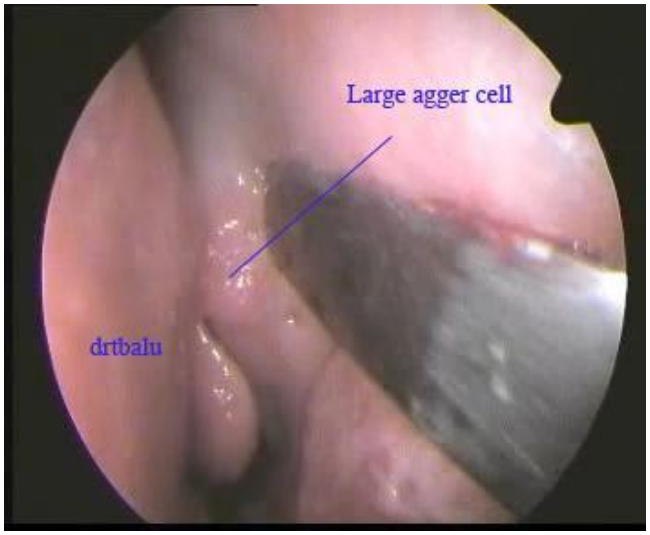
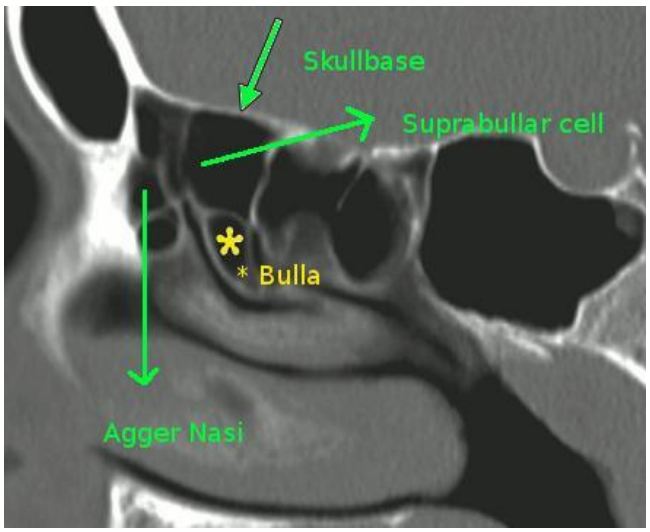


Fig. 5: Large aggernasi cell



Saggittal view of CT nose and pns showing large agger nasi cell and supra bullar cell. Note frontal recess is bounded anteriorly by agger nasi cell and posteriorly by suprabullar air cell

Supraorbital ethmoidal cells:

These cells are also known as suprabullar cells. These cells belong to the anterior ethmoidal group. These cells originate from immediatly behind the frontal recess and extends over the orbit due to

pneumatization of orbital plate of frontal bone. This in association with large agger nasi can compromise frontal sinus drainage pathway. During surgery this air cell can be mistaken as frontal sinus. On transillumination with telescope the light will be seen in the inner canthal area as compared to that of supra orbtial area which will be transilluminated by frontal sinus[8].

Middle turbinate pneumatization:

Sometimes middle turbinate may be pneumatized. A pneumatized middle turbinate is known as concha bullosa. Pneumatization of concha bullosa usually arises from frontal recess area / agger nasi air cells. This should be considered as a normal variant and does not require surgery. Ofcourse extensive pneumatization may narrow osteomeatal complex impeding sinus drainage. This condition requries surgical intervention [7].

Interlamellar cell of Grunwald:

This cell is pneumatization of vertical portion / lamella of middle turbinate. This pneumatization arises from the superior meatus. This air cell was first described by Grunwald [7]**.**

Onodi Cell:

This cell is also known as sphenoethmoidal cell. This cell is christened after Adolf Onodi [9]of Budapest who studied the relationship of ethmoid aircells in relation to optic nerve. These cells belong to posterior ethmoid group. Onodi cells extend superiorly and laterally to the sphenoid sinus. Its pneumatization can reach up to the clinoid process. This cell is related to optic nerve in its lateral wall. If this cell is large carotid artery could be seen bulging from its posterior wall. Any attempt to open the sphenoid sinus through this cell is fraught with dangerous complications like injury to optic nerve / internal carotid artery. Pre op CT scan is the best way to identify this condition. “Forewarned is forearmed”.

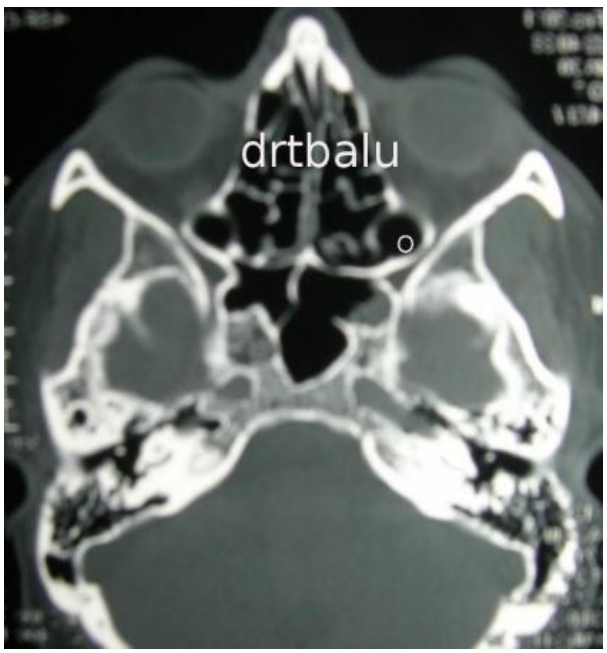


Fig. 6: CT scan axial cut showing onodi cell

Frontal sinus:

Among the para nasal sinuses this sinus shows the maximum variations. Infact variations are so immense that it can safely be stated that frontal sinuses are unique in each and every individual. It may be absent in 5 % of individuals. It is more or less shaped like a L. Drainage channel of frontal

sinus is highly variable.

Posterior wall: corresponds to the anterior wall of the anterior cranial fossa.

Floor: is formed by the upper part of the orbits.

Frontal sinus appear very late in life. Infact they are not seen in skull films before the age of 6.

The sinus drains into the anterior part of the middle meatus through the fronto nasal duct.

Frontal outflow tract showsconglomeratizationof air cells

Types of frontal sinus air cells include:

I – Type I frontal cell (a single air cell above agger nasi)

II – Type II frontal cell (a series of air cells above agger nasi but below the orbital roof)

III – Type III frontal cell (this cell extends into the frontal sinus but is contiguous with agger nasi cell)

IV – Type IV frontal cell lies completely within the frontal sinus

Sphenoid sinus:

Is located in the skull base at the junction of the anterior and middle cranial fossa. Pnematisation of sphenoid starts during the 4th year of childhood and gets completed by the 17th year. The sphnoid sinuses vary in size and may be asymmetric. Each of these sinuses are separated by an intersinus septum which may not be in the midline. Because of asymetry the intersinus septum could be deviated to one side. This intersinus septum could attach posteriorly to the bony carotid canal. Care should be taken while removing this septum without injuring carotid artery canal. It is prudent to use true cut forceps to remove the intersinus septum. Pneumatization of sphenoid sinus can also involve anterior clinoid process, posterior clinoid process and posterior end of nasal septum.

They drain through the superior meatus via a small ostium about 4mm in diameter located disadvantageously 20mm above the sinus floor.

This sinus is related to several important vital structures. They are:

1. Pituitary gland lies above the sphenoid sinus.

2. Optic nerve and internal carotid arteries traverse its lateral wall.

3. The nerve of pterygoid canal lie in the floor of the sinus.

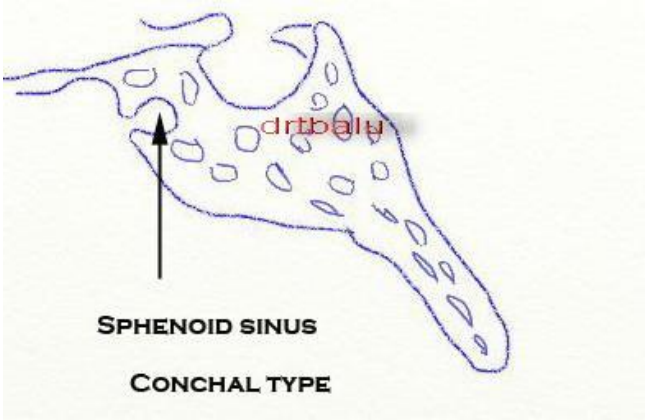
Hence infections of sphenoid sinus may involve the optic nerve if the canal of the optic nerve is dehiscent.

Types of sphenoidal sinuses depending on extent of pneumatization:

Conchal type:

In this type the area below the sella is a solid block of bone without an air cavity. This type is common in children under the age of 12 because pneumatization begins only after the age of 12.

Fig. 7: Conchal type pneumatization of sphenoid



Presellar type:

In this type the air cavity does not penetrate beyond the coronal plane defined by the anterior sellar wall.

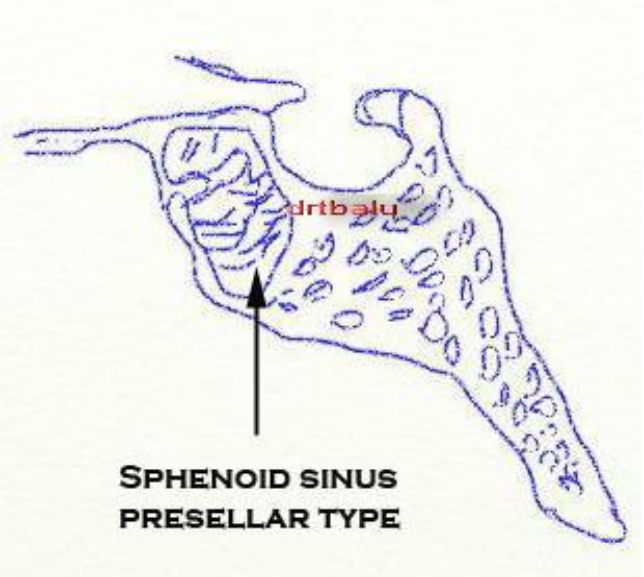


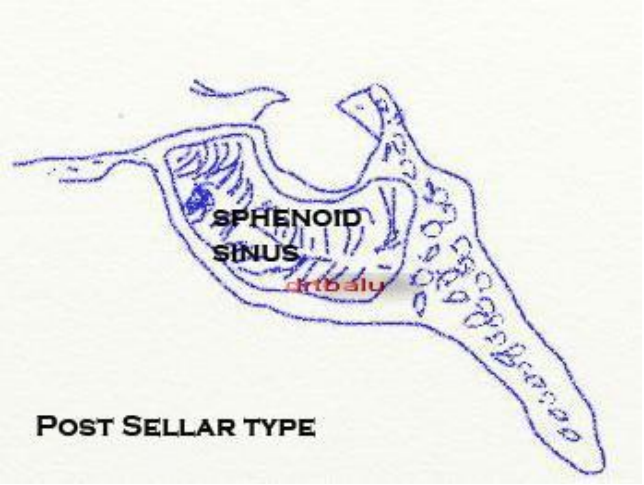
Fig. 8: Sellar type pneumatization

Sellar type:

In this type the air cavity extends into the body of the sphenoid below the sella and may extend as far posteriorly as the clivus. This type is commonly seen in 85% of individuals.

In well pneumatized sphenoid sinus, the pterygoid canal and a segment of maxillary division of trigeminal nerve could be identified in the lateral recess of the sphenoid sinus.

Fig. 9: Post sellar type pneumatization



The roof of the sphenoid (planum sphenoidale) anteriorly is continuous with the roof of ethmoidal sinus. At the junction of the roof and posterior wall of sphenoid the bone is thickened to form the tuberculum sella. Inferior to the tuberculum sella on the posterior wall is the sella turcica. It forms a bulge in the midline. The bone over the sella could be 0.5 – 1 mm thick. This may get thinner inferiorly. It is hence easy to breech the sella in this tinnest part. This area can be easily identified by a bluish tinge of the dura which is visible through the thin bony covering.

Possible variations of intersinus septum are as follows:

1. A single midline intersinus septum extending on to the anterior wall of sella.

2. Multiple incomplete septae may be seen

3. Accessory septa may be present. These could be seen terminating on to the carotid canal or optic nerve.

Lateral wall of sphenoid sinus: is related to the cavernous sinus. This sinus is formed by splitting of the dura. It extends from the orbital apex to the posterior clinoid process. Cavernous sinus contains very delicate venous channels, cavernous part of internal carotid artery, 3rd, 4th and 6th cranial nerves. It also contains some amount of fatty tissue.

Fig. 10: Endoscopic view of interior of sphenoid sinus

The prominence of internal carotid artery is the postero lateral aspect of the lateral wall of sphenoid sinus. This prominence can be well identified in pneumatized sphenoid bones. On the antero superior aspect of the lateral wall of sphenoid sinus is seen the bulge formed by the underlying optic nerve. These two prominences are separated by a small dimple known as the opticocarotid recess. The optic nerve and internal carotid artery is separated from the sphenoid sinus by a very thin piece of bone. Bone dehiscence is also common in this area.

Histology:

All these sinuses are lined by respiratory type pseudo stratified ciliated columnar epithelium. This epithelium is composed of 4 major types of cells:

Ciliated columnar cells

Nonciliated columnar cells

Goblet cells

Basal cells

Since the mucosa lining the paranasal sinuses are attached to the bone it is known as mucoperiosteum. The mucoperiosteal lining of the sinus is thinner than the mucosal lining of the nasal cavity. This lining is continuous with the mucosal lining of the nasal cavity via the sinus ostium [6]. The ostium is though which various sinus cavities communicate with the nasal cavity. The concept of muciliary clearance mechanism pushing the secretions out via the natural ostium is the concept behind functional endoscopic sinus surgery.

References

1. [Article in German] Feldmann H. HNO-Klinik, Universität Münster. Laryngorhinootologie. 1998 Oct;77(10):587-95.

2. Leonardo da Vinci. 1977. Anatomical drawings from the Royal Collection. London: Royal Academy of Arts. P 50.

3. Blanton PL, Biggs NL (1969) Eighteen hundred years of controversy: the paranasal sinuses. Am J Anat 124(2):135– 47

4. Stammberger H (1989) History of rhinology: anatomy of the paranasal sinuses. Rhinology 27(3):197–210.

5. Bolger WE, Anatomy of the Paranasal Sinuses. In: Kennedy DW, Bolger WE, Zinreich J (2001) Diseases of the sinuses, Diagnosis and management. B.C. Decker.

6. Van Cauwenberge P, Sys L, De Belder T et al (2004) Anatomy and physiology of the nose and the paranasal sinuses. Immunol Allergy Clin North Am 24(1):1–17

7. Stammberger HR, Kennedy DW (1995) Paranasal sinuses: anatomic terminology and nomenclature. The Anatomic Terminology Group. Ann Otol Rhinol Laryngol Suppl Oct;167:7–16

8. Polavaram R, Devaiah AK, Sakai O et al (2004) Anatomic variants and pearls-functional endoscopic sinus surgery. Otolaryngol Clin North Am 37(2):221–42.

9. Kantarci M, Karasen RM, Alper F et al (2004) Remarkable anatomic variations in paranasal sinus

region and their clinical importance. Eur J Radiol 50(3):296–302.

Choanal atresia

August 30, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Abstract: Choana is also known as posterior nasal aperture. Nasal airway continues with that of posterior nares. Air from nasal cavity finds its way into the lungs via the choanal apertures. In some children the choana may be congenitally closed. This causes either total (bilateral choanal atresia) or partial (unilateral choanal atreisa) nasal obstruction. Child being obligate nasal breathers, find it rather difficult to breathe when there is bilateral choanal atresia. This is more so during the first 6 weeks of life. Hence bilateral choanal atresia should be considered as an emergency in paediatric age group. This article attempts to discuss the etiopathology and management of this condition.

Introduction:

Choanal atresia is actually a developmental failure of the nasal cavity to communicate with nasopharynx.. This condition is rather rare occurring in about 1 in 10000 live births. This condition is more common in female children the ratio being 2:1 [1]. About 50% of of these patients have other associated congenital anamolies. The most commonly associated congenital anomaly is CHARGE Syndrome.

CHARGE Syndrome include [2]:

C- Coloboma

H- Heart disease

A- Choanal atresia

R- Mental and growth retardation

G- Genital hypoplasia

E- Ear deformities

Other anomalies associated with choanal atresia include [3]:

1. Polydactyly
2. Nasal / auricular deformities
3. Palatal deformities
4. Down’s syndrome
5. DiGeorge syndrome
6. Meningocele
7. Menigoencephalocele

8. Treacher Collin's syndrome

9.Mid face hypoplasia

Types of choanal atresia:

1. Unilateral / Bilateral : Unilateral choanal atresia is an incidental finding and is verycommon (about 70%) [4]. High degree of suspicion is necessary to identify this condition. An

infant with unilateral choanal atresia will have problems suckling milk from the breast opposite to the side of atresia.

2. Bony atresia – 90%, Membranous atresia – 6%, Combined atreisa 4% [5].

Anatomic deformitites associated with choanal atresia include [6]:

1. The bony atretic plate is situated infront of the posterior bony septum

2. Nasal cavity is narrow in these patients

3. Lateral pterygoid plates are found to be thickened compromising the nasal airway

4. Medially vomer is thickened

5. Inbetween lateral pterygoid and vomer is the membranous plate

6. High arched palate is common in these patients

History of choanal atresia:

This condition was first described by Johann George Roederer in 1755. This was later termed as an anatomical abnormality of palatine bone by Adolf Otto in 1854 [7].

Carl Emmert has been successfully credited with the first choanal atresia repair in 1854 [7].

Embryology of choanal atresia:

Development of face and cranial structures occur during the first 12 weeks of gestation. The development of choanae takes place between the 4th and 11th weeks of gestation [8]. Cranial

structures develop from neural crest cell migration. Development of nose begins during the 4th week of gestation. This is indicated by the formation of nasal pits. During the 5th week of gestation the nasal pits begin to fold inwards intothe mesenchyme forming nasal sacs. These primitive nasal sacs are separated from oral cavity by oronasal membranes. During the 8th week of gestation this oronasal membrane ruptures creating nasal cavity and a primitive choana located at the junction of nasal cavities and nasopharynx. During this phase of development there is gradual proliferation of neural crest cells. These cells contribute to the formation of skull base and nasal vaults. During the 10th week of gestation the nasal septum and developing palate fuse. The

primitive choanae gets pushed posteriorly. This choanae which forms during the 10th week of gestation is known as "Secondary choanae". In normal foetus these secondary choanae are patent

for a functioning airway between the anterior nasal cavity and nasopharynx [9].

Theories of development of choanal atresia [10]:

Four theories for the development of choanal atresia:

1. Persistence of a buccopharyngeal membrane from the foregut.

2. Persistence of the nasobuccal membrane of Hochstetter – most commonly accepted theory.

3. The abnormal persistence or location of mesodermal adhesions in the choanal region.

4. A misdirection of mesodermal flow secondary to local genetic factors better explains the popular theory of persistent nasobuccal membrane

Other theories that are not so widely accepted include:

1. Resorption of the floor of secondary nasal fossa

2. Incomplete dorsal extension of nasal cavity

3. Migration of dorsal part of fronto nasal process to fuse with the palatal shelves

Studies have revealed that cranio facial anamolies with mesenchymal damage and cell disruption were found in mothers who ingested high doses of vitamin A during their period of pregnancy.

This has been attributed to disturbances in migration pattern of neural crest cells, which is also followed by disturbances in mesoderm development in the cranio facial area [11].

In patients with choanal atresia the atretic plate has the following boundaries:

Superior – under surface of body of sphenoid

Lateral – medial pterygoid lamina

Medial – vomer

Inferior – horizontal plate of palatine bone

This anatomical knowledge of atrophic plate is highly valuable while performing surgery on these patients.

Clinical features:

Bilateral choanal atresia is considered to be a neonatal emergency. These infants present with asphyxia neonatorum. Bilateral choanal atresia is commonly associated with:

1. Nursing difficulties – Sucking difficulties

2. Respiratory distress – Cyclic. When the infant falls asleep it becomes breathless as the nose is blocked

3. Respiratory infections – can occur due to aspiration

4. Recurrent nasal allergies

5. Cyanosis which gets better when the child cries.

6. Cry is not normal (Rhinolalia clausa)

7. Bilateral choanal atresia is also commonly associated with other birth defects like orofacial defects, cardiac defects and limb defects [12].

Teratogenic syndromes causing bilateral choanal atresia include:

1. Methimazole embryopathy [13]

2. Carbimazole embryopathy [14]

Detailed drug intake history is a must in diagnosing embryopathies.

Unilateral choanal atresia is commonly missed. These infants find difficulty in sucking milk frombreast opposite to the side of choanal atresia. These children have unilateral nasal obstruction with nasal discharge. A strong degree of suspicion is a must to identify this condition.

Tests to identify choanal atresia:

1. Attempting to pass 6-8 sized French plastic catheter through the nose. If there is no atresia the catheter will effortlessly pass through the nasal cavity into the nasopharynx. If there is choanal atresia then a typical solid feeling would be encountered at about 3-3.5 cms from the alar rim. If obstruction is encountered within 1-2 cms from the anterior nares, then it could be caused due to traumatic deflection of nasal septum due to trauma.

2. When a wisp of cotton is placed closed to the nasal opening then it would move in the presence of air flow.

3. When methylene dye is instilled in to the anterior nasal cavity it can be seen passing through the nasopharynx. Obstruction due to choanal atresia will prevent flow of methylene dye into the nasopharynx.

CT scan imaging is virtually diagnostic:

It has the unique advantage of differentiating membranous choanal atresia from bony ones. In patients with combined atresia it will also reveal the contribution of these two elements to the atretic plate. Actual structures involved in the atretic plate would be clearly seen. In all these patients

vomer appears to be thickened, the lateral nasal wall bends medially to fuse with vomer thereby obstructing the nasal cavity.

Management:

In bilateral choanal atresia securing the air way takes the top priority. An oral airway can be introduced to tide over the immediate crisis.

Role of intraoral nipple:

A nipple with a large opening (McGovern) Nipple [15]can be introduced into the oral cavity of the infant to tide over the crisis. This provides adequate airway to the infant. A small infant feeding tube can be passed through another small opening present in the nipple or alongside the nipple for

gavage feeding. This helps to buy time till the child has gained adequate weight to withstand corrective surgery.

Tracheostomy:

This should be considered only on rarest of rare occasions when the patient is not able to adequately maintain oral airway.

Timing of repair in unilateral choanal atresia:

Choanal atresia repair in unilateral atresia is delayed till the child reaches its first birthday. This allows

the surgery site to enlarge thereby reducing the risk of post op stenosis. Bleeding is also reduced if surgery is delayed. Older infants tolerate stenting better than young ones.

Trans nasal approach:

Transnasal approach: (using endoscopes): The surgery is performed under general anesthesia. A self retaining nasal speculum is used to expose the nasal cavity and the atretic plate. If the atresia is membranous in nature a simple perforation of the same under endoscopic guidance would suffice.

The nasal cavity is decongested using 4% xylocaine with adrenaline in the concentration of 1 in 10,000 concentration. Under endoscopic guidance a mucosal incision is made and the mucosal flaps are elevated exposing the posterior vomer and lateral pterygoid lamina. A diamond burr on an angled hand piece is used to drill the atretic bony plate. It is perforated at the junction of the hard palate and the vomer. Incidentally this is the thinnest part of the atretic plate. This procedure was first described by Stankiewicz. To improve visualisation the inferior turbinate can be out fractured

or even be trimmed. After drilling care is taken to preserve the mucosal flaps. A silastic stent is placed into each nostril passing through the drilled neo choana. This helps in reducing the incidence of restenosis. Stent is kept in place for atleast 6 weeks 19.

Opening made should be large enough to allow smooth passage of suction catheter. 3-4 size Endotracheal tube can be used as stent to prevent restenosis. The size should be choosen carefully in such a way that it should be adequate to prevent restensosis and inadequate to cause nasal

regurgitation.

Caution:

While performing this procedure caution must be taken not to injure the sphenopalatine vessels behind the middle turbinate.

Advantages of this procedure:

1. This process is faster and easier

2. Blood loss is minimal

3. Can be performed in children of all ages who do not have associated external nasal deformities

4. Child can be immediately breast fed

5. Child can be discharged on the 3rd day itself

Disadvantages:

1. Vision is highly limited especially in the new born

2.Inability to adequately remove enough of the posterior vomerine septal bone and prevent restenosis

3. Longer stenting time

4. Endoscopes do not offer binocular vision

5. Cannot be done safely and with good results on patients with multiple nasal and nasopharyngeal

anomalies.

First transnasal repair of choanal atresia was performed by Dehaen[18] in 1985. He used microscope for magnification and visulization of the atretic plate.

Endoscopic transnasal approach[16] is facilitated by advances in instrumentation, anesthesia, imaging etc. CT imaging reveals the amount of contribution to the stenotic segment by the lateral nasal wall. It is prudent to avoid drilling too much into the lateral nasal wall as it could damage the

sphenopalatine vessels[17]. Use of powered instrumentation like soft tissue shavers and drills have made this procedure a lot safer.

Use of Mitomycin C[19] to prevent restenosis:

In addition to routine stenting restenosis can be prevented by topical application of Mitomycin -C, which is an antimetabolite known to inhibit fibroblast formation.

Complications of use of stents following transnasal repair of choanal atresia:

1. Formation of granulation tissue

2. Crust formation

3. Septal perforation

4. Persistent nasal discharge

Transpalatal approach:

This approach is more suitable for bilateral choanal atresia. Under general anesthesia the palate is exposed with a mouth gag. Palatal mucosa is infiltrated with 2% xylocaine with 1 in 100000 adrenaline. This infiltration serves the dual purpose of helping in flap elevation and providing much needed hemostasis during the entire surgical procedure. A curved incision is given on the palate starting from just behind the maxillary tuberosity on one side and is carried medially along

the alveolar ridge up to the canine region. The same incision is carried out even on the opposite side. A “U” shaped palatal flap is elevated. This flap is elevated up to the edge of the hard palate.

The greater palatine neurovascular bundle is preserved at all costs. The soft palate is now retracted posteriorly and superiorly exposing the posterior edge of hard palate. This area is the area for dissection. The posterior edge of hard palate is taken down using kerrison’s punch or drill. The

nasal mucosa is exposed. This mucosal flap is lifted posteriorly till the choana is reached. The posterior portion of nasal septum and lateral superior nasal wall is also taken down. A stent is placed. The mucosal flap is then redraped in position.

Complications of transeptal approach:

1. Pressure necrosis of columella

2. Plugging of stent

3. Displacement of stent

4. Palatal dehiscence

5. Maxillary hypoplasia causing malocclusion

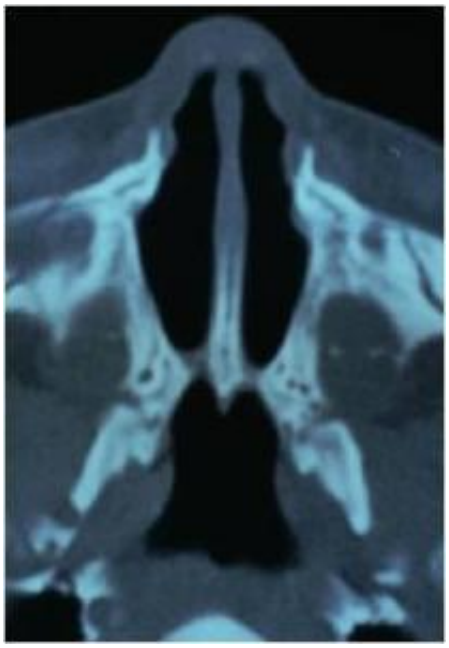
6. Granulation tissue formation around the stents.

Conclusion:

The main aim of choanal atresia repair is maintenance of nasal airway at all costs.

Success of choanal atresia surgery is determined by the necessity of post op dilatation of the chonal orifice or revision surgery on the same patient due to dense re stenosis.

Endoscopic view of choanal atresia



CT scan showing bilateral choanal atresia

References

1. Theogaraj SD, Hoehn JG, Hagan KF. Practical management of congenital choanal atresia. Plast Reconstr Surg 1983; 72: 634-42.

2. Bergstrom L, Owens O. Posterior choanal atresia: a syndromal disorder. Laryngoscope 1984; 94: 1273-6.

3. Harris J, Robert E, Kallen B. Epidemiology of choanal atresia with special reference to the

CHARGE association. Pediatrics 1997; 99: 363-7.

4. Dobrowski JM, Grundfast KM, Rosenbaum KN, Zajtchuk JT. Otorhinolaryngic manifestations of CHARGE association. Otolaryngol Head Neck Surg 1985; 93: 798-803.

5. Brown OE, Pownell P, Manning SC. Choanal atresia: a new anatomic classification and clinical management applications. Laryngoscope 1996; 106: 97-101.

6. Harner SG, McDonald TJ, Reese DF. The anatomy of congenital choanal atresia. Otolaryngol Head Neck Surg 1981; 89: 7-9.

7. Hengerer AS, Brickman TM, Jeyakumar A. Choanal atresia: embryologic analysis and evolution of treatment, a 30-year experience. Laryngoscope. May 2008;118(5):862-866.

8. Dunham ME, Miller RP. Bilateral choanal atresia associated with malformation of the anterior skull base: embryogenesis and clinical implications. Ann Otol Rhinol Laryngol. Nov 1992;101(11):916-919.

9. Johnston MC. The neural crest in abnormalities of the face and brain. Birth Defects Orig Artic Ser. 1975;11(7):1-18.

10. http://www.drtbalu.co.in/choa_atre.html

11. Mossey PA, Little J, Munger RG, Dixon MJ, Shaw WC. Cleft lip and palate. Lancet. Nov 21 2009;374(9703):1773-1785.

12. Burrow TA, Saal HM, de Alarcon A, Martin LJ, Cotton RT, Hopkin RJ. Characterization of congenital anomalies in individuals with choanal atresia. Arch Otolaryngol Head Neck Surg. Jun 2009;135(6):543-547

13. Clementi M, Di Gianantonio E, Pelo E, Mammi I, Basile RT, Tenconi R. Methimazole embryopathy: delineation of the phenotype. Am J Med Genet. Mar 5 1999;83(1):43-46.

14. Wolf D, Foulds N, Daya H. Antenatal carbimazole and choanal atresia: a new embryopathy. Arch Otolaryngol Head Neck Surg. Sep 2006;132(9):1009-1011.

15. McGovern, FH. Association of congenital choanal atresia and congenital heart disease. Reprot of two cases. Ann. Otol.Rhin and laryng 1953; 62:894-895

16. McGovern, FH. Association of congenital choanal atresia and congenital heart disease. Reprot of two cases. Ann. Otol.Rhin and laryng 1953; 62:894-895

17. Pasquini E, Sciarretta V, Saggese D, Cantaroni C, Macri G, Farneti G. Endoscopic treatment of congenital choanal atresia. Int J Pediatr Otorhinolaryngol 2003; 67: 271-6.

18. Dehaen F, Clement PA (1985) Endonasal surgical treatment of bilateral choanal atresia under optic control in the infant. J Otolaryngol 14: 95-98.

19. Selik YK, Biesman BS, Rebeiz EE (2000) Topical application of Mitomycin-C in endoscopic dacryocystorhinostomy. Am J Rhinol 14: 205-207.

Complications of sinusitis

January 16, 2013 · Rhinology

Authors

Balasubramanian Thiagarajan

Abstract

Anatomically paranasal sinuses are in close proximity to vital structures like brain and orbit. Lesions affecting nasal sinuses can affect these areas as well. Infections involving mucosal lining of paranasal sinuses can also spread to these adjacent vital areas. This article attempts to study the orbital and intra cranial complications of sinusitis.

Introduction:

Anatomically paranasal sinuses are closely related to orbit and skull base. They share common bony boundaries, and blood supply[1]. Complications attributed to sinusitis are caused by spread of infections to adjacent areas[2].

Routes of spread:

1. Bacterial infections from the sinuses can spread through natural dehiscences and weakness of the bony barriers. In chronic infections the surrounding bone undergoes sclerosis, while in acute sinusitis massive osteolysis is commonly seen.

2. Lamina papyracea is a paper thin bone separating the orbit from the ethmoidal sinuses. Congenital dehiscences of this bone is commonly seen through which spread of infection can occur from the ethmoids into the orbit. In childhood the frontal sinuses are underdeveloped and orbital complications are caused commonly by acute ethmoiditis.

3. Floor of the frontal sinuses form the roof of the orbit. In older children and in adults frontal sinus infections can spread into the orbit causing orbital complications.

4. Infraorbital canal in the floor of the orbit is a weak area through which infections from orbit may enter into the maxillary sinus.

5. Spread of infection can also occur via diploic veins present in the frontal bone. These veins are known as the veins of Breschet. This is preceded by thrombophlebitis.

6. Venous connections between the sinuses and the orbit donot have any valves facilitating spread of infection from the sinuses to the orbit[3].

7. The roots of the second premolar and the first upper molar are intimately related to the floor of the maxillary sinus. This facilitates a two way spread of infection. In cases of isolated maxillary sinusitis dental causes must be suspected.

Predisposing factors for developement of complications following sinusitis:

1. Immunocompromised patient (e.g. HIV)

2. Diabetes mellitus

3. Irregular treatment for sinus infections

4. Inappropriate / Inadequate antibiotic therapy

Complications of sinusitis include:

1. Orbital complications (Commonest)

2. Intracranial complications [4](including meningitis, subdural empyema, intracerebral abscess, epidural abscess, cavernous sinus thrombosis)

3. Mucocele

4. Pyocele

5. Osteomyelitis

6. Pyocele

7. Facial cellulitis

8. Subperiosteal abscess

Orbital complications of sinusitis:

This is more common in younger individuals. Orbital complications are frequently caused by ethmoiditis because ethmoidal sinus shares its border with orbit. Lamina papyracea (paper thin bone) is the lateral barrier separating ethmoidal sinus from orbit. This bone can easily be breached during active infections. Ethmoiditis as a cause for orbital complication is rather common in young children, where as in adults frontal sinusitis happens to be the most common cause for orbital complications. In adults sphenoid sinusitis can involve optic nerve leading on to blindness. Left orbit is commonlyinvolved than right one [5]. The incidence of orbital complications following sinusitis is highly variable i.e. More than 20%.[7]

Hubert's classification of orbital complications of sinusitis[8]:

Hubert classified orbital complications arising from sinusitis into five groups:

Group I: Inflammatory oedema of eyelids with or without oedema of orbital contents.

Group II: Subperiosteal abscess with oedema of lids or spread of pus to the lids.

Group III: Abscess of orbital tissues

Group IV: Mild to severe orbital cellulitis with phlebitis of ophthamic veins

Group V: Cavernous sinus thrombosis

Smith & Spencer classification of orbital complications:

Group I: Preseptal cellulitis – Characterised by oedema of eyelids without tenderness, visual loss or limitation of ocular mobility.

Group II: Orbital cellulitis without abscess formation – characterised by diffuse oedema of adipose tissues of orbit.

Group III: Orbital cellulitis with subperiosteal abscess formation with displacement of the globe. May

or may not be associated with visual loss. Ocular mobility is restricted.

Group IV: Orbital cellulitis with intraperiosteal abscess. Here the displacement of globe is severe with restriction of ocular mobility.

Group V: Cavernous sinus thrombosis.

Route of spread to orbit:

1. Through bony dehiscence / defect (may be congenital or acquired)

2. Through neurovascular foramen

3. Through venous channels

Chandler classified orbital infection into 5 stages [9]:

Stage I:

Periorbital cellulitis. Also known as preseptal cellulitis. It should not be confused with orbital cellulitis which occurs behind the orbital septum. This condition is actually inflammation and infection of the eyelid and portions of skin around the eye. These patients donot have proptosis, limitation of eye movement, painful eyemovement and loss of vision. These features actually help in differentiating preseptal cellulitis from orbital cellulitis. Eyelids in these patients appear swollen but not tender. There is no chemosis.

Stage II:

Orbital cellulitis. Also known as post septal cellulitis. This condition is characterized by pronounced oedema and inflammation of orbital contents without abscess formation. These patients have varying degrees of proptosis, restriction of ocular movements, painful eye movements and chemosis. Since loss of vision could occur in these patients it is mandatory to monitor their vision on a regular basis.

Stage III:

Subperiosteal abscess. Abscess develops in these patients between bone and periosteum. Orbital contents are invariably displaced in an infero lateral direction due to mass effect of accumulating pus. Chemosis and proptosis areinvariablypresent. Decreased ocular mobility and loss of vision may also occur in these patients.

Stage IV:

Orbital abscess. This involves collection of pus within the orbital contents. This is caused due to relentless progression of orbital cellulitis or rupture of orbital abscess. These patients have severe proptosis, complete opthalmoplegia and commonly loss of vision also.

Stage V:

Cavernous sinus thrombosis. Development of bilateral ocular signs is the classic feature of this stage. This stage carries worse prognosis. Features of this stage include [10]:

1. Fever

2. Head ache

3. Photophobia

4. Proptosis

5. Opthalmoplegia

6. Loss of vision

7. Cranial nerve palsies (3, 4, V1, V2, and 6th nerves)

References

1. Bolger WE, Anatomy of the Paranasal Sinuses. In: Kennedy DW, Bolger WE, Zinreich J (2001) Diseases of the sinuses, Diagnosis and management. B.C. Decker.

2. Balasubramanian Thiagarajan. Anatomy of Paranasal sinuses [Internet]. Version 9. Ent Scholar. 2012 Nov 8. Available from: http://entscholar.wordpress.com/article/anatomy-of-paranasal-sinuses/.

3. Younis RT, Lazar RH, Anand VK. Intracranial complications of sinusitis: a 15-year review of 39 cases. Ear Nose Throat J 2002; 81:636-44.

4. Bluestone CD, Steiner RE, Intracranial complications of acute frontal sinusitis. South Med J 1965; 58:1-10.

5. Gamble R.C (1933), Acute inflammation of the orbit in children. Archives of Ophthalmology 10, 483-497.

6. Chandler.J.R, Langenbrunner.D.J Stevens.E.R (1970). The pathogenesis of Orbital complications in acute sinusitis- Laryngoscope 80, 1414-1428

7. Chandler.J.R, Langenbrunner.D.J Stevens.E.R (1970). The pathogenesis of Orbital complications in acute sinusitis- Laryngoscope 80, 1414-1428

8. Lavania A , Sharma V ,Reddy N S , Baksh R .Orbital cellulitis: A Complication of sinusitis; Kathmandu University medical journal 2005 Jul;3(3):292-293

9. Chandler JR, Langenbrunner DJ, Stevens ER. The pathogenesis of orbital complications in acute sinusitis. Laryngoscope 1970; 80:1414-28.

10. Yarington CT, Jr. Cavernous sinus thrombosis revisited. Proc Royal Soc Med. 1977; 70: 456-459.

Concept of unified airway

September 2, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Abstract:

According to European rhinological society guidelines chronic rhinosinusitis with nasal polypi and chronic rhinosinusitis without nasal polypi are two different entities. This article attempts to review published literature which attempts to study link between nasal polyposis and lower airway disorders. The concept of unified airway attempts precisely to explain this linkage. Developmentally and functionally it makes sense to combine both upper and lower airways in studying pathophysiology of various airway disorders.

Introduction:

An attempt to define the following terminologies will not be out of place.

Rhinosinusitis / Nasal polyposis:

Number of authors have attempted to define this condition, majority of these definitions were based on symptomatology and duration of the disease. Till date there is no universally accepted definition of this condition [1]. European rhinological society has stepped in to define rhinosinusitis in unambiguous terms.

Rhinosinusitis / Nasal polyposis is defined as:

Inflammation of mucosal lining of nose and paranasal sinuses characterised by two or more symptoms, one of which should be either nasal block / nasal obstruction / nasal discharge (anterior / posterior discharge):

Presence or absence of facial pain / tenderness

Reduction / Loss of smell.

And / either:

Endoscopic signs of :

Polypi

Mucopurulent discharge / oedematous mucosa over middle meatus

And / either

CT scan changes showing mucosal oedema / OMC obstruction

The severity of the disease can be classified using visual analogue scale:

1. Mild – Visual analogue score 0-3

2. Moderate – Visual analogue score 3-7

3. Severe – Visual analogue score – 7-10

Visual analogue score of more than 5 affects quality of life of the patient.

Duration of the disease:

Acute : < 12 weeks with complete resolution of symptoms

Chronic: > 12 weeks without complete resolution of symptoms. Chronic rhinosinusitis can show periods of acute exacerbations.

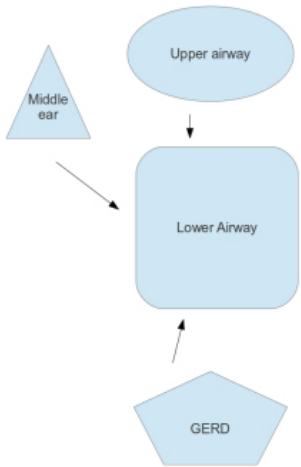
Studies attempt to divide chronic rhinosinusitis with / without nasal polyposis based on inflammatory markers [2], [3]. Why nasal mucosa balloons in to polypi in certain patients with chronic sinusitis is still an enigma. In these patients polypi tend to recur even after apparantly complete surgical removal. Some studies have demonstrated pronounced eosinophilia and IL – 5 expression in patients with nasal polyposis than in those with chronic sinusitis without nasal polypi. Samter in his classic description states the presence of a triad in some of these patients. This triad goes after his name “Samter’s Triad”. Patients with Samter’s triad have asthma, aspirin hypersensitivity and nasal polyposis.

Majority of patients with chronic rhinosinusitis with nasal polypolis manifest with asthma and bronchial hyper-responsiveness 3. The role of medico-surgical management of nasal polyposis in managing patients with associated lower airway symptomatology is still not clear. Some studies even go to the extent of suggesting that surgical removal of nasal polypi in these patients actually worsens bronchial asthma in these patients [4]. Vleming [5]in his study has reported that nasal polypectomy actually helped in alleviating symptoms due to bronchial asthma in these patients.

Theoretically larynx is considered to be the boundary between the upper and lower airway system. Functionally speaking the major function of the upper air way is to conduct air and also make it fit for gas exchange in the lower airway by adding moisture to inspired air. The lower air way plays a vital role in gas exchange. Currently the term unified airway encompasses the following structures: middle ear mucosa, nasal cavity and paranasal sinus mucosa, and the entire tracheo bronchial tree. Studies have demonstrated that pathologies affecting one portion of this unified airway ultimately progresses to involve the other areas as well.

It was Keller in 1920 [6]who noticed that commonly patients with lower respiratory diseases also had complaints pertaining to the upper airway as well. Keller also hypothesised that decreased ability of upper air way to humidify inspired air leads to worsening of bronchial asthma.

Concept of unified airway



Current status is that whenever a diagnosis of rhinitis or asthma is considered then the entire airway should be

evaluated [7]. Even Gastro oesophageal reflux [8]disease has also been implicated with airway disorders. Hence no investigation for asthma / upper airway disorder is complete without upper GI endoscopy.

Criteria supporting the concept of unified airway:

1. Patients with upper airway diseases like rhinitis and rhinosinusitis have increased prevalence of lower respiratory disorders like bronchial asthma

2. Patients with lower airway disorders like bronchial asthma have increased prevalence of rhinosinusitis

3. Pathophysiological mechanisms causing both upper and lower airway disorders are more or less similar

4. Treating one component of unified airway disorder should have beneficial effect on the other components as well.

Corren's[9] role in strengthening the concept of unified airway:

Corrent in his classical paper clearly demonstrated the coexistance of rhinitis in patients with bronchial asthma. He also observed a temporal relationship between bronchial asthma and rhinitis. He stated that attacks of bronchial asthma was preceded by an attack of rhinitis.

According to Guerra et al [10]sufferers of allergic rhinitis are three times more likely to develop asthma when compared to controls in their study.

Starting from 1991 when Natioal Institute of Health [11]classified asthma as inflammatory disease, thereby shifting the focus from bronchospasm to inflammation. This caused a major shift in the treatment stratergy. Inflammation is the common denominator defining both upper and lower airway disorders. It should also be stressed at this point that the lining epithelium of both upper and lower airway i.e ciliated columnar epithelium functionally are similar. In response to chronic inflammation the

mucosal lining of both upper and lower airway demonstrate similar histopathological patterns i.e. Inflammatory cellular infiltrate and eventual thickening of basement membrane. Eosinophils have been identified as the common inflammatory cell causing problems in disorders involving both upper and lower airway. Other inflammatory cells that have been implicated in unified airway disorders include CD4 T-Lymphocytes and mononuclear cells.

The most compelling proof of the concept of unified airway comes from the fact that the inflammatory mediators released starting the cascade of inflammation in the upper and lower airway are virtually the same. Inflammatory triggers from one portion of airway also affects other portions as well.

Hence by viewing the entire airway as a single unit patient reaps the benefit of unified treatment protocol. It is always prudent to control chronic sinusitis in addition to managing asthma by bronchodilators. Successful management of chronic rhinosinusitis results in decreased asthma medication[12] in these patients with tell tale improvement in pulmonary function. An additional benefit being reduced number of exacerbations.

Mechanisms responsible for unified airway disease:

1. Nasobronchial reflex

2. Loss of nasal protection to lower airway

3. Shared inflammation through out the respiratory tract

Nasobronchial reflex:

This concept was first proposed by Sluder[13] who stated that nasal irritation can cause bronchial irritation leading on to bronchospasm. Kaufmann[14] and Wright added further proof to this concept. In their study they applied silica particles to the nasal mucosa of individuals without bronchial asthma and demonstrated there is increased lower airway resistance following this application. Even in individuals in whom this reflex is not readily demonstrable delayed changes have been demonstrated in the lungs starting from 30 mins – 4 hours following antigenic challenge of nasal mucosa. This observation suggests that other mechanisms in addition to direct reflex arc[15] could be responsible for interaction between upper and lower airway structures.

Loss of nasal protection to nasal airway:

This concept was first proposed by Shturman – Ellestein in 1978 [16]. In their classic experiments performed on volunteers they were able to demonstrate that mouth breathing worsened bronchospasm, while nasal breathing reduced lower airway resistance. This phenomenon can be explained when the primary function of the nosal airway (airconditioning) is taken into consideration. Lower air way responds to unconditioned inspired air by increasing their resistance to the inspired air. This can infact be considered to be a protective mechanism.

Shared inflammation:

This concept is also catching up as one of the explanations for common pathological basis of unified airway disorders. This concept has found its root from the observation that inflammatory disorders of various portions of the unified airway are caused by release of same immune / inflammatory mediators. Braunstahl et al [17]observed that stimulation of one portion of the airway mucosa with anigen results in a system wide inflammatory changes within hours.

Conclusion:

The concept of unified airway disorders has infact shifted the focus from individually managing various disorders affecting the components of airway to that of unified management modality. This calls for multidisciplinary approach in managing these patients in an optimal manner. Specialities involved in designing management protocol for unified airway disorders include Thoracic medicine and otolaryngologists.

References

1. Anon JB, Jacobs MR, Poole MD, Ambrose PG, Benninger MS, Hadley JA, et al. Antimicrobial treatment guidelines for acute bacterial rhinosinusitis. Otolaryngol Head Neck Surg. 2004 Jan;130(1 Suppl):1-45.

2. Hedman J, Kaprio J, Poussa T, Nieminen MM. Prevalence of asthma, aspirin intolerance, nasal polyposis and chronic obstructive pulmonary disease in a population-based study. Int J Epidemiol. 1999;28(4):717-22.

3. Larsen K. The clinical relationship of nasal polyps to asthma. Allergy Asthma Proc. 1996;17(5):243-9.

4. Lawson W. (1991) The intranasal ethmoidectomy: an experience with 1077 procedures. 101:367–371

5. Vleming M., Stoop A. E., Middelweerd R. J., de Vries N.(1991) Results of endoscopic sinus surgery for nasal polyps. 5:173–176.

6. Keller I. Sinus disease. Lancet 1920;40:133–4

7. Bousquet J, van Cauwenberge P, Khaltaev N, et al. Allergic rhinitis and its impact on asthma (ARIA): executive summary of the workshop report. Allergy 2002;57:841–55.

8. Loehrl TA, Smith TL. Chronic sinusitis and gastroesophageal reflux: are they related? Curr Opin Otolaryngol Head Neck Surg 2004;12:18–20.

9. Corren J. The connection between allergic rhinitis and bronchial asthma. Curr Opin Pulm Med 2007;13:13–8.

10. Guerra S, Sherrill DL, Martinez FD, et al. Rhinitis as an independent risk factor for adult-onset asthma. J A llergy Clin Immunol 2002;1 09;41 9-25.

11. National A sthma Education and Prev ention Program (NA EPP). Guidelines for the diagnosis and management of asthma. National Heart, Lung, and Blood Institute. National A sthma Education Program. Ex pert Panel Report. J A llergy Clin Immunol 1 991 ;88:425-534.

12. Stelmach R, do Patrocinio T, Nunes M, et al. Effect of treating allergic rhinitis with corticosteroids in patients with mild to moderate persistent asthma. Chest 2005;1 28:31 40-7 .

13. Sluder G. Asthma as a nasal reflex. JAMA 1919;73:589–91.

14. Kaufman J, Wright GW. The effect of nasal and nasopharyngeal irritation on airway resistance in man. Am Rev Respir Dis 1969;100:626–30.

15. 15. Assanasen P, Baroody FM, Abbott DJ, et al. Natural and induced allergic responses increase the ability of the nose to warm and humidify air. J Allergy Clin Immunol 2000;106:1045–52.

16. Shturman-Ellstein R, Zeballos RJ, Buckley JM, et al. The beneficial effect of nasal breathing on exercise-induced bronchoconstriction. Am Rev Respir Dis 1978;118:65–73.

17. Braunstahl GJ, Kleinjan A, Overbeek SE, et al. Segmental bronchial provocation induces nasal inflammation in allergic rhinitis patients. Am J Respir Crit Care Med 2000;161:2051–7.

Dentigerous cyst

From supernumerary teeth

October 3, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Abstract

Dentigerous cysts are the most common developmental odontogenic cysts. They are usually derived from the epithelial remnants of tooth forming organs. These cysts increase in size gradually. There may also be associated bone resorption. Managing these lesions creates problems in children. It is always better to be conservative in managing this problem in children because dentition is yet to complete in them.

Introduction:

Dentigerous cysts are the most common developmental odontogenic cysts. They are usually derived from the epithelial remnants of tooth forming organs[1]. Dentigerous cysts are classically defined as cystic lesions that are caused by separation of follicles from around the crown of unerupted teeth. Most commonly dentigerous cyst involves lower 3rd molar (mandibular) [2]. Dentigerous cysts were earlier termed as “Follicular cysts” since it was assumed that these cysts were derived from tooth follicle which is a mesodermal structure. Later this term was abandoned as it was conceived on an erroneous perception. Dentigerous cysts can also be caused by:

1. Impacted teeth

2. Supernumerary teeth – Is defined 8 as teeth in excess of usual configuration of 20 deciduous and 32 permanent teeth. Dentigerous cysts arising from supernumerary teeth accounts for nearly 5% of all these cysts.

3. Ectopic teeth (eruption of a teeth in sites other than the natural position). Most commonly seen ectopically erupted teeth involves 3rd molars

4. Rarely a tooth / root of teeth may be found in the sinus cavity. This teeth may have dentigerous cyst associated with it [3].

Theories of dentigerous cyst formation:

Usually all dentigerous cysts arise from the enamel organ after completion of amelogenesis. Dentigerous cyst arises due to accumulation of fluid causing separation of enamel of the unerupted tooth. The fluid present inside the cyst is hyperosmolar due to the presence of albumin, immunoglobulin and squamous epithelial debris. This hyperosmolar fluid causes influx of extracellular fluid into the cyst causing huge expansion of cyst to occur. The epithelial lining of the cyst secretes collagenase and osteoclast activating factor which causes local bone resorption causing further increase in the size of the cyst. This enlarging cyst encloses the crown of the unerupted teeth and is

attached to its cemento-enamel junction.

Theories explaining genesis of Dentigerous cyst:

1. Theory of stimulation

2. Theory of inflammation[4]

Incidence:

Studies reveal that dentigerous cyst constitute more than a quarter of all jaw cysts. It predominates during the 2nd – 3rd decades of life[5]. There is a very slight male preponderance.

Majority of dentigerous cysts involves the mandibular third molar while maxillary canine is the next in the order of involvement. Very rarely dentigerous cyst can occur from ectopically erupted tooth within the maxillary sinus[6].

Symptoms:

These patients usually present with painless slow growing swelling involving the affected area. This swelling is very frim on palpation indicating cortical expansion. If it is present in the upper jaw then the swelling could involve the hard palate also.

These cysts are usually painless and dormant. There may be some degree of expansion of cortical bone. Presence of pain and rapid swelling definitly indicates inflammation. Fistula can rarely occur when the dentigerous cyst is present in the maxillary sinus. These patients usually present with evidence of sinusitis[7]. When these cysts are aspirated then yellowish fluid 9 could be observed. The swelling may also reduce in size following aspiration only to increase in size later.

Histology:

Histopathological examination of the cyst wall showed the cyst to be lined by reduced enamel epithelium. Connective tissue stroma will show features of primitive type of ectomesenchyme. Findings would depend on whether there is inflammatory component to the cyst is present or not. In non infected cysts the lining epithelium is 2-4 layers thick formed by primitive ectomesenchyme. These lining cells are low cuboidal to columnar. Retepegs could be seen only in cysts which are infected. The connective tissue stroma is loose and is rich in acid mucopolysaccharides. When the dentigerous cyst isinflamedthen it is characterised by the presence of hyperplastic rete ridges and the cyst wall demonstrates inflammatory infiltrate.

Theories of dentigerous cyst formation:

Intrafollicular theory:

According to this theory cyst formation occurs due to fluid accumulation between the layers of inner and outer enamel epithelium after crown formation.

Enamel hypoplasia theory:

This theory suggests that dentigerous cyst formation occurs due to degeneration of stellate reticulum at a very early stage of tooth development. There is also associated enamel hypoplasia.

Main’s theory:

This theory suggests that impacted tooth exerts pressure on the follicle with resulting obstruction of venous outflow. This induces rapid transudation of fluid across the capillary walls. This causes an

increase in the hydrostatic pressure exerted causing separation of crown from the follicle. This may be associated with reduced enamel epithelium.

Radiographic features:

In plain radiographs these cysts present as a well defined unilocular radiolucency. Often there is a demarcating sclerotic border. Since the cyst lining is derived from reduced enamel epithelium this

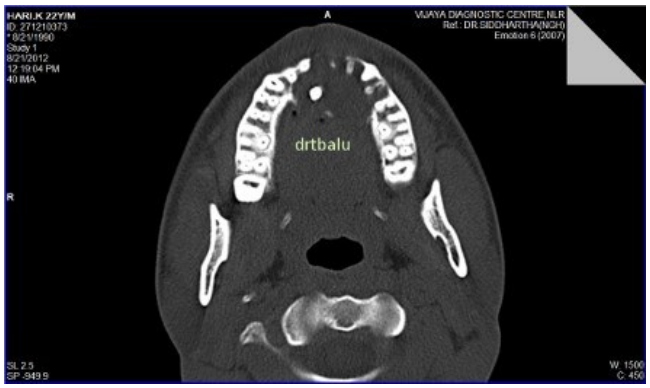
radiolucency preferentially surrounds the crown of the teeth. A large dentigerous cyst may provide an impression as if it is multilocular. This appearance is due to the persistence of bony trabeculae within the radiolucency. These cysts are particulary unilocular in nature.

Radiographic types of dentigerous cysts:

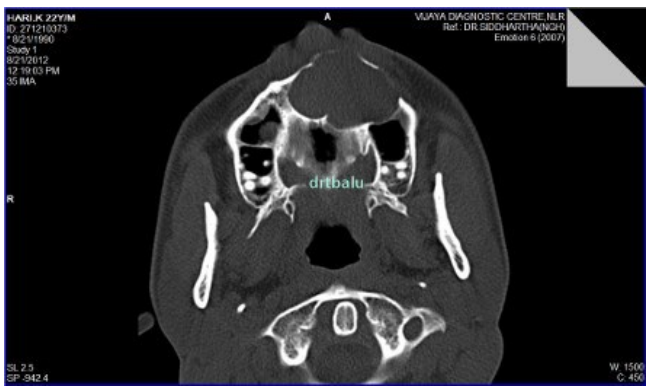
1. Central variety: In this variety the radiolucency surrounds the crown of the unerupted teeth. The crown can clearly be seen projecting into the cyst lumen.

2. Lateral variety: In this variety the cyst develops laterally along the tooth root, partially encircling the crown

3. Circumferential variety: The cyst entirely surrounds the unerupted teeth. Radiologically the unerupted teeth could be seen within the cyst cavity.



Dentigerous cyst showing unerupted teeth



CT scan showing dentigerous cyst

Treatment:

The usual treatment of dentigerous cyst is careful enucleation of the cyst in toto. Unerupted tooth if present should usually be removed along with the cyst. Sometimes if eruption of this teeth is Sometimes orthodontic treatment may be advocated to assist eruption of unerupted teeth.

Dentigerous cyst

Marsupialization:

This is more conservative method than enucleation of the entire cyst. This should be considered as the first line of management in children with dentigerous cyst. Major advantage of this procedure is loss of viable permanent tooth buds can be prevented. These patients should be followed up carefully by performing radiological imaging every 6 months in order to keep an eye on potential recurrence. This follow up should be continued atleast for a period of 2 years following marsupialization.

Unerupted teeth seen inside dentigerous cyst

Why dentigerous cysts should be treated?

1. They block eruption of normal teeth

2. They increase in size and cause displacement of teeth

3. They can cause bone destruction

4. They can cause displacement of vital structures like inferior alveolar nerve

References

1. Amin ZA., Amran M., Khairudin. Removal of extensive maxillary dentigerous cyst via a Caldwell-Luc procedure. Arch Orofac Sci 2008;3(2):48-51.

2. Neville BW, Dam DD, Allen CM, Bouquet JE. Oral and Maxillofacial Pathology. 2nd ed. Saunders; c2001. 768p

3. Srinivasa PT, Sujatha G, Thanvir MN, Rajesh P. Dentigerous cyst associated with an ectopic third molar in the maxillary sinus: A rare entity. Indian J Dent Res 2007:18(3):141-3

4. Soon Jae Hwang, Heung-Man Lee, Dae Hyung Kim, Dong Jin Lee, Jang Hyeog Lee, Jin Ho Choi, Sang Hag Lee. Dentigerous Cyst Involving the Maxillary Sinus. J Rhinol November 2001;8(1,2):54-7.

5. Mervin S and Paul SM. Cysts of the Oral Cavity and Maxillofacial Regions. 4th ed. Blackwell Munksgaard Publishers; c2007. 228p.

6. Soon Jae Hwang, Heung-Man Lee, Dae Hyung Kim, Dong Jin Lee, Jang Hyeog Lee, Jin Ho Choi, Sang Hag Lee. Dentigerous Cyst Involving the Maxillary Sinus. J Rhinol November 2001;8(1,2):54-7.

7. Albert SP. Dentigerous Cyst in Maxillary Sinus. J Natl Med Assoc 1970;62(6):453-4.

Drug induced gingival overgrowth

September 5, 2012 · Laryngology

Authors

Balasubramanian Thiagarajan

Abstract:

Gingival hyperplasia / hypertrophy is a rather common condition. This article reviews literature pertaining only to gingival overgrowth following drug ingestion. A wide range of causes have been attributed to gingival overgrowth. Drug induced overgrowth commonly occurs following medications prescribed for non dental causes. Pathogenesis of gingival overgrowth following ingestion of certain drugs is still unsure. Certain high risk co existant factors like presence of gingivitis has been implicated. Management of this condition should take into consideration the condition for which the offending drug has been prescribed. Physicians should be aware of drugs that could cause gingival overgrowth in order to identify and manage this problem.

Introduction:

A large number of drugs have been implicated as cause for gingival hypertrophy / hyperplasia. Use of the term hypertrophy / hyperplasia is rather controversial. These terms do not accurately reflect the current understanding of the current histopathological scenario. Gingival enlargement in these patients is not due to increase in the number of periodontal cells but due to an increase in the extracellular volume [1]. This increase in the extracellular volume is caused by hyperplasia involving fibroblasts.

Gingival overgrowth was first described in dental literature of 1960′s[2] when case reports started appearing about children who developed enlarged gingiva due to treatment of epilepsy using phenytoin. This condition was also described in children born of epileptic mothers who were being treated with sodium valproate. These children were branded to be suffering from fetal valproate syndrome [3]. Fetal valproate syndrome was first described by Di Liberti in 1984 [4]. These children also had neurodevelopmental problems. Associated congenital defects in this syndrome include Neural tube defects, congenital heart defects, orofacial clefts, and limb defects.

Drugs involved in causing gingival enlargement:

Three types of drug categories [6]have been implicated as causative factors of gingival enlargement. They include:

1. Anti epileptic drugs – Phenytoin, Phenobarbitone, Valproic acid, Primidone, Vigabatrin and carbamazepine.

2. Calcium channel blockers – Nifidepine, Verapamil, Diltiazem and Amlodepin

3. Immunosuppresive drugs – Cyclosporine

Studies reveal that drug induced gingival overgrowth usually occurs within first three months of starting drug therapy with the offending drug. This usually begins as an enlargement involving Interdental papillae.

Prevalance:

Review of literature shows a wide variation in prevalance rates [7]. A high figure of 50% has been quoted for the drug phenytoin, where as for cyclosporin it is 30% and for calcium channel blockers 10%. Recent studies have demonstrated the synergistic effect of cyclosporin with calcium channel blockers in causing gingival overgrowth [8]. Indian statistics are rather sketchy in this aspect. One study reports that 57% of epileptic children in the age group 8-13 on phenytoin developed gingival overgrowth within the first 3 months of starting the treatment[9].

Etiology:

Etiology although enough pointers are there to point fingers at an offending drug, is still considered to be multifactorial. The relationship between the drug dosage, duration of therapy and sex prediliction is still not clear. There is still a raging debate going on as to whether drug induced gingival hyperplasia could be caused by hyperplasia of gum epithelium or of subcutaneous connective tissue or both.

Certain predisposing risk factors have been indentified and documented. They include:

Poor oral hygiene: Presence of dental plaque can provide a reservoir for accumulation of drugs like phenytoin / cyclosporin[8].

In patients who have undergone orthodontic procedures the presence of nickel could predispose to formation of gingival over growth

Susceptibility of some subpopulation of fibroblasts and keratinocytes to phenytoin, cyclosporin and other drugs which could cause gingival overgrowth

Number of langhans cells[10]present in the oral epithelium is another risk factor. More the number worse the risk. These drugs have a tendency to accumulate inside these cells causing prolonged effect on the gums.

Cytochrome P-450 gene polymorphism [11]can cause individual variations in drug metabolism predisposing to gingival overgrowth

In patients using calcium channel blockers gingival overgrowth could be caused by:

Defective collagenase activity

Blockage of aldosterone synthesis from adrenal cortex followed by feedback increase in the secretion of ACTH

Upregulation of keratinocytes growth factor

Upregulation of transforming growth factor alpha[412]

The tissue overgrowth is classically of dense collagen tissue and other connective tissue elements. Scattered inflammatory cells can also be seen. Classically gingival tissue adjacent to anterior teeth is more commonly affected than the posterior ones. Presence of bacterial plaque in the teeth is essential for gingival overgrowth to occur. Histology of overgrowth revealed hyperplasia of connctive tissue, epithelial acanthosis and elongated rete ridges.

Picture showing gingival overgrowth

Management:

Stopping / substituting the offending medicine. Gingival overgrowth reverts back to normal within 3 months.

Maintenance of strict oral hygiene

Regular mouth wash using cholorhexidine

Oral metronidazole for 21 days

Surgical removal of the gingival overgrowth (gingivectomy)

References

1. Desai P, Silver JG. Drug induced gingival enlargement. J Can Dent Assoc 1998; 64: 263-8.

2. Kimbal OP. The treatment of epilepsy with sodium diphenyl hydantoinate. J Am Med Assoc 1939; 112: 1244-5.

3. Rodriguaz-Vazquez M, Carrascosa-Romero MC, Parddal-Fernandoz M, et al. Congenital gingival hyperplasia in a neonate with foetal valproate syndrome Neuropediatrics 2007; 38: 251-2.

4. DiLiberti JH, Farndon PA, Dennis NR, Curry CJ. The fetal valproate syndrome. Am J Med Genet 1984;19:473-481

5. Perlik F, Kolinova M, Zvarova J, et al. Phenytoin as a risk factor in gingival hyperplasia. Therap Drug Monitor 1995; 17: 445-8.

6. Perlik F, Kolinova M, Zvarova J, et al. Phenytoin as a risk factor in gingival hyperplasia. Therap Drug Monitor 1995; 17: 445-8.

7. 6. Ellis JS, Seymour RA, Steele JG, et al. Prevalence of gingival overgrowth induced by calcium channel blockers: a community based study. J Periodontol. 1999; 70: 63-7.

8. Spratt H, Boomer S, Irwin CR, et al. Cyclosporin associated gingival overgrowth in renal transplant recipients. Oral Dis 1999; 5 : 27- 31.

9. Mejia LM, Lozada-Nur F. Drug induced gingival hyperplasia. E medicins . Medscope. com 2009; article / 1076264

10. Kinane DF, Drummond JR, Chisholm DM. Langerhans cells in human chronic gingivitis and phenytoin induced gingival hyperplasia. Arch Oral Biol 1990; 35: 561-4.

11. Seymour RA, Ellis JS, Thomason JM. Risk factors for drug induced gingival overgrowth . J Clin Periodontol. 2000; 27 : 217-23.

12. Lafzi A, Farahani RMZ, Shoja MM. Amlodipine induced gingival hyperplasia. Oral Med Pathol Oral Cir Buccal 2006; 11: E480-2.

Epistaxis

October 7, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Introduction:

Epistaxis is defined as bleeding from the nasal cavity. It is actually a Greek word for nose bleed. It is actually a very common problem and hence its incidence is rather difficult to access. Crude estimates or its incidence ranges from 5 – 14%[1]. The incidence of epistaxis also shows significant increase during winter months / hot dry climates with low humidity. This climatic increase in incidence of epistaxis has been attributed to increase in the incidence of upper respiratory tract infections. Forceful blowing of inflammated nasal mucosa provokes epistaxis in these patients[2]. Classically epistaxis is known to manifest bimodal incidence – with peaks in age groups of 2-10 and 60-80. Only a small percentage of this population seek Otolaryngologist intervention. This amounts to about 1% of all patients with nasal bleed[3].

History:

Epistaxis has been a centre of all folklores. Some associated epistaxis with “Love” while others believed that it foretold death / some form of severe illness. On the whole spirits were believed to cause epistaxis. Lupton in 1601 suggested that the patients use their own blood from epistaxis to write the words “consummatum est” on the forehead in order to avoid further episodes. These words were uttered by Jesus Christ as he was dying on the cross. The exact meaning of these words mean “Its finished”. Moncrief in 1716 fried the patient’s own epistaxis blood and applied the same as snuff as treatment. Hippocrates one of the earliest physicians appreciated that pressure on alae nasi in patients with epistaxis managed to stop the nasal bleed. Ali Ibn Rabban Al-Tabiri [4]a Persian Hakim (850 A.D.) wrote in his classic treatise that epistaxis was due to swelling of vein (Retro columellar) and its eventual rupture. Giovanni Battisata Morgagni an Italian Anatomist observed turgid blood vessels located about a finger’s breadth from the anterior nasal cavity. He attributed epistaxis to bleeding from this area.

Carl Michel, James Lawrence Little and Kiesselbach identified venous plexus over the anterior part of cartilagenous septum as a source of epistaxis. In 1868 Pilz performed the first documented common carotid artery ligation as treatment of epistaxis. Alfred Seiffert in 1928 introduced the concept of ligation of internal maxillary artery via trans antral approach as a treatment modality for epistaxis. It was Henry Goodyear in 1937 who first ligated anterior ethmoidal artery to treat epistaxis.

Vascularity of nasal mucosa:

Nasal mucosa is highly vascular. The submucosal blood vessels of nasal cavity receive blood supply from both interanal and external carotid systems. The general rule of thumb is that the area of nasal cavity below the level of middle turbinate is supplied by external carotid branches while the area above the level of middle turbinate [5]is supplied by internal carotid artery. Anastomosis between these two systems are known to occur within the nasal cavity. It should be borne in mind

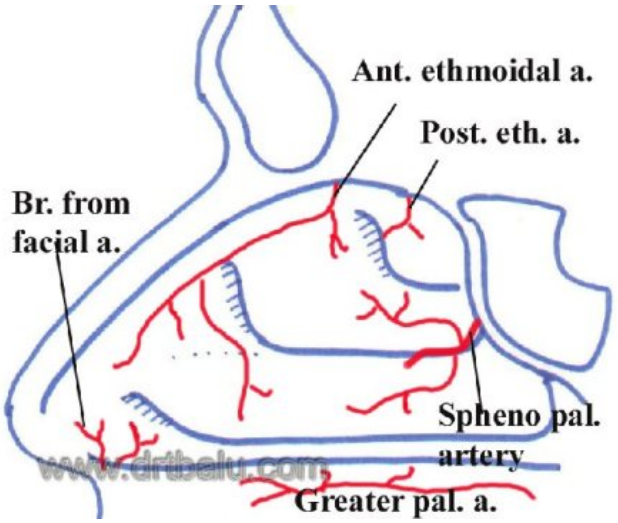
that the pressure levels at the internal carotid artery is higher than that of external carotid artery.

External carotid system: Blood from the external carotid system reaches the nasal cavity via the facial and the internal maxillary arteries which are branches of the external carotid artery. The artery of epistaxis is the sphenopalatine branch of internal maxillary artery. This is called so because this vessels supplies the major portion of the nasal cavity. It enters the nasal cavity at the posterior end of the middle turbinate to supply the lateral nasal wall, it also gives off a septal branch which supplies the nasal septum.

Facial artery: the superior labial branch of the facial artery is one of its terminal branches. It supplies the anterior nasal floor and anterior portion of the nasal septum through its septal branch.

Internal maxillary artery: after entering into the pterygopalatine fossa this vessel gives rise to[6] branches. These branches are posterior superior aleveolar artery, descending palatine artery, infra orbital artery, sphenopalatine artery, pterygoid artery, and pharyngeal artery. The descending palatine artery enters the nasal cavity through the greater palatine canal to supply the lateral wall of the nose, it also contributes blood supply to the nasal septum through its septal branch.

Internal carotid system: the internal carotid artery supplies the nasal cavity via its ophthalmic artery. It enters the orbit via the superior orbital fissure and divides into many branches. The posterior ethmoid artery one of the branches of ophthalmic artery exits the orbit via the posterior ethmoidal foramen located 2-9 mm anterior to the optic canal. The anterior ethmoidal artery which is larger leaves the orbit through the anterior ethmoidal foramen. Both these vessels cross the roof of the ethmoid and descends into the nasal cavity through the cribriform plate. It is here that these vessels divide into lateral and septal branches to supply the nose.

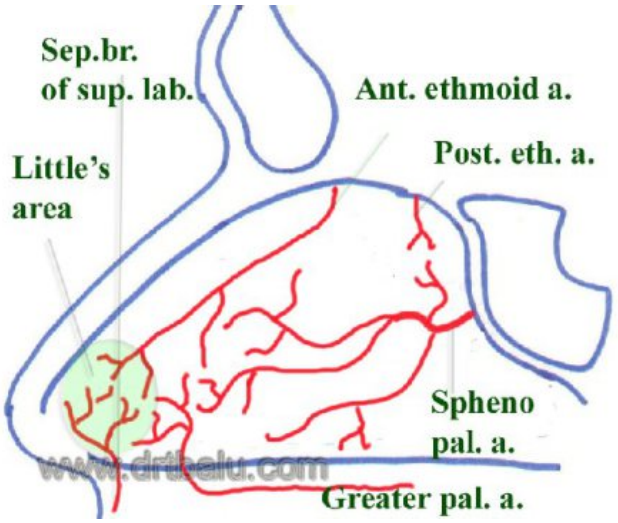


Blood supply of lateral nasal wall

Little's area: This area is located in the anterior part of the cartilagenous portion of the nasal septum. Here there is extensive submucous anastomosis of blood vessels both from the external and the internal carotid systems. Bleeding commonly occurs from this area since it is highly vascular and isalso exposed to the exterior. Anastomosis occur between the septal branches of sphenopalatine artery, greater palatine artery, superior labial artery and the anterior ethmoidal artery. This plexus is also known as Keisselbach's plexus. Bleeding from this area is common because mucosal drying occurs commonly here and this area is easily accessible to nose picking. Among the vessels taking part in the anastomosis the anterior ethmoidal artery is from the internal carotid system while the other vessels are from the external carotid system. Bleeding from this area is clearly seen and easily accessible and flows through the anterior nasal cavity hence it is known as anterior bleed.

Two areas have been implicated in epistaxis. Little's area has been implicated in anterior epistaxis and Woodruff's plexus in posterior epistaxis. Anterior epistaxis is common in children while posterior epistaxis is common in adults.

Woodruff's plexus: is responsible for posterior bleeds. This area is located over the posterior end of the middle turbinate. The anastomosis here is made up of branches from the internal maxillary artery namely its sphenopalatine and ascending pharyngeal branches. The maxillary sinus ostium forms the dividing line between the anterior and posterior nasal bleeds. Posterior nasal bleeds are difficult to treat because bleeding area is not easily accessible. Bleeding from Woodruff's plexus commonly occur in patients with extremely high blood pressure. Infact this plexus acts as a safety valve in reducing the blood pressure in these patients, lest they will bleed intracranially causing more problems. In patients with posterior bleeds it is difficult to access the amount of blood loss because most of the blood is swallowed by the patient.



Little's area

Woodruff in 1949 reported a group of large blood vessels in the lateral wall of inferior meatus posteriorly. He was able to visualize these blood vessels using a rigid nasopharyngoscope. He coined the term "Naso nasopharyngeal plexus" to describe these vessels. He suspected the association between the presence of these dilated blood vessels and posterior epistaxis. He was not sure whether these vessels are veins or arteries.

Microdissection studies revealed a superficial collection of fragile fairly large calibre blood vessels lying just beneath the surface mucosa [7]. There was very little intervening connective tissue. Histological studies revealed that the epithelium overlying the posterior inferior meatus was typical respiratory epithelium. The blood vessels in this area were sinus like with very little muscle or fibrous tissue within their walls. The average blood vessel diameter in this area is 1-2mm.

Shaheen described Woodruff's plexus as an arterial plexus formed by anastomosis between pharyngeal, posterior nasal, sphenopalatine and posterior septal arteries. Microdissection and histological studies have proved Woodruff's plexus to be venous in origin.

Bleeding from the blood vessels of Woodruff's plexus could result in a slow but prolonged ooze. Since these blood vessels have no muscle walls, hemostasis is poor. Post nasal packing will have to be resorted to in rare cases to stop bleeding.

Etiology: The etiology of epistaxis is not just simple or straight forward. It is commonly multifactorial, needing careful history taking and physical examination skill to identify the cause. For purposes of

clear understanding the etiology of epistaxis can be classified under two broad heads, i.e. local and systemic causes [7].

Local factors causing epistaxis: include vascular anamolies, infections and inflammatory states of the nasal cavity, trauma, iatrogenic injuries, neoplasms and foreign bodies. Among these causes the commonest local factors involved in epistaxis is infection and inflammation. Infections and inflammation of the nasal mucous membrane may damage the mucosa leading on to bleeding from the underlying exposed plexus of blood vessels. Chronic granulomatous lesions like rhinosporidiosis can cause extensive epistaxis.

Aneurysms involving the internal carotid artery may occur following head injury, injury sustained during surgical procedures. These extradural aneuryms and aneurysms involving the cavernous sinus may extend into the sphenoid sinus wait for the opportune moment to rupture. It can cause sudden fatal epistaxis, or blindness. Urgent embolisation is the preferred mode of management of this condition.

Trauma is one of the common local causes of epistaxis. It is commonly caused by the act of nose picking in the Little's area of the nose. This is commonly seen in young children. Acute facial trauma may also lead to epistaxis. Patients undergoing nasal surgeries may have temporary episodes of epistaxis.

Irritation of the nasal mucous membrane: any disruption of normal nasal physiology can cause intense drying and irritation to the nasal mucosa causing epistaxis. These episodes are common during extremes of temperature when the nasal mucosa is stressed to perform its airconditioning role of the inspired air. In these conditions there is extensive drying of nasal mucosa causes oedema of the nasal mucous membrane. This oedema is caused due to venous stasis. Ultimately the mucosa breaches exposing the underlying plexus of blood vessels casuing epistaxis.

Anatomical abnormalities: Common anatomical abnormality causing epistaxis is gross septal deviation. Gross deviations of nasal septum causes disruption to the normal nasal airflow. This disruption leads to dessication / drying of the local mucosa. The dry mucosa cracks and bleeds.

Septal perforations: Chronic non healing septal perforations can cause bleeding from the granulation tissue around the perforation.

Neoplasms: involving the nose and paranasal sinuses can cause epistaxis. Neoplasms include benign vascular tumors like hemangioma, juvenile nasopharyngeal angiofibroma, and malignant

neoplasms like squamous cell carcinoma. If epistaxis occurs along with secretory otitis media then nasopharyngeal carcinoma should be the prime suspect.

Systemic causes for epistaxis:

Hypertension is one of the common systemic causes of epistaxis. Accumulation of atheroscerotic plaques in the blood vessels of these patients replaces the muscular wall. This replacement of muscular wall reduces the ability of the blood vessels to constrict facilitating epistaxis. This is one of the common causes of posterior nasal bleeds. It commonly arises from the Woodruff's plexus found close the posterior end of the middle turbinate. Beran etal concluded that hypertension had no significant impact as etiological factor for epistaxis [8].

Hereditary hemarrhagic telengectasia also known as Osler – Rendu – Weber disease is another systemic disorder known to affect the blood vessels of the nose. This is an autosomal dominant non

sex linked disorder. This disease causes loss of contractile elements within the blood vessels causing dilated venules, capillaries and small arteriovenous malformations known as telengectasia. These changes can occur in the skin, mucosal lining the whole of the respiratory passage and urogenital passage. Bleeding from these telengectasia is difficult to control. Bleeding invariably starts when the patient reaches puberty. Common cause of mortality in these patients is gastrointestinal bleed.

This condition is more common in women 5:1. Serious epistaxis is known to occur in nearly 80% of these patients by the age of 30 [10]. Depending on the amount of blood loss decision on transfusion is made. Nasal packing and cauterization is advocated for mild to moderate bleeding in these patients. Treatment of this condition is rather palliative since the underlying disease is not curable [9]. Laser coagulation of bleeders have been tried out with reasonable degree of success in these patients. Topical oestrogen therapy following laser cauterization of bleeders help in squamous metaplasia of nasal epithelium thereby reducing the incidence of bleeding 10. In very retractable cases nasal obliteration too has been attempted (Young's procedure).

Systemic diseases like syphilis, tuberculosis & wegner's granulomatosis cause epistaxis because of their propensity to cause ulceration of the nasal mucous membrane. Viral infections like dengue and haemorrhagic fever cause epistaxis due to reduced platelet count.

Blood dyscrasias can also cause epistaxis. A low platelet count is one common cause of nasal bleed in this category. In thrombocytopenia the platelet count is less than 1 lakh. Epistaxis can start when the platelet count reduces to 50,000. Platelet deficiency can be caused by ingestion of drugs like aspirin, indomethacin etc. Hyperspenism can cause thrombocytopenia in idiopathic thrombocytopenic purpura. These patients need to be transfused fresh blood in adequate quantities. Only when thee platelet count increases will the nasal bleed stop.

Incidence: The incidence of epistaxis is known to be slightly higher in males. It also has a bimodal distribution affecting young children and old people.

Evaluation: While evaluating a patient with epistaxis it is absolutely necessary to assess the quantum of blood loss. The blood pressure and pulse rate of these patients must be constantly monitored. These patients will have tachycardia. Infusion of fluid must be started immediatly.

Initially ringer lactate solution will suffice. If the patient has suffered blood loss of more than 30% of their blood volume (about 1.5 liters) then blood transfusion becomes a must. Further examination should be started only after the patient's general condition stabilises.

History: Careful history taking is a must. History taking should cover the following points:

1. History regarding the freqency, severity and side of the nasal bleed.

2. Aggravating and relieving factors must be carefully sought.

3. History of drug intaken must be sought.

4. History of systemic disorders like hypertension and diabetes mellitus must be sought.

Physical examination:

The nasal pack if any must be removed. Anterior nasal examination should be done, first attempted without the use of nasal decongestants. If visualisation is difficult due to oedema of the nasal mucosa then nasal decongestants can be used to shrink the nasal mucosa. The solution used for anesthetising the decongesting the nose is a mixture of 4% xylocaine and xylometazoline.

Nasal endoscopy can be performed under local anesthesia to localise posterior bleeds.

Investigations:

If bleeding is minimal no investigation is necessary.

If bleeding is more then a complete blood work up to rule out blood dyscrasias is a must. It includes bleeding time, clotting time, platelet count and partial thromboplastin time.

Imaging studies like CT scan of the para nasal sinuses must be done to rule out local nasal conditions of epistaxis. Imaging must be done only after 24 hours of removing the nasal packing. Scans done with the nasal pack or immediatly after removing the nasal pack may not be informative.

In diffiucult and intractable cases angiography can be done and the internal maxillary artery can be embolised in the same sitting. This procedure should be reserved only for cases of intractable nasal bleeding.

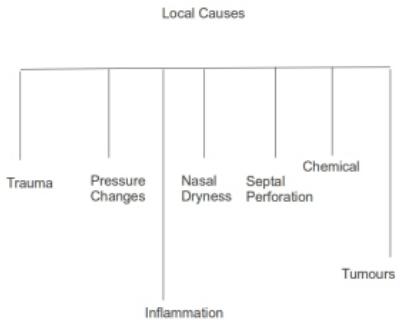
Classification of etiological factors of epistaxis:

For better understanding etiological factors of epistaxis has been classified under two heads:

1. Local causes

2. Systemic causes

Local cause of epistaxis



Trauma:

This is the most common cause of epistaxis. This category includes:

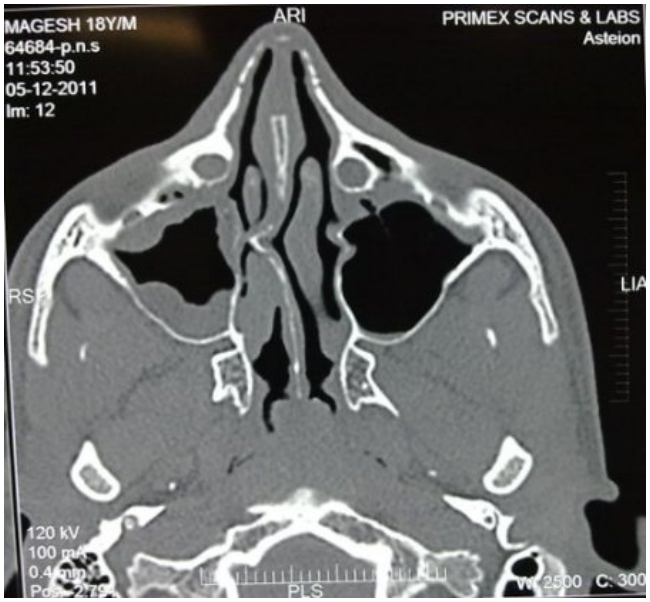
Fractures

Self induced digital trauma / foreign body

Iatrogenic – surgical procedures involving nose and sinus and skull base

Barometric changes – Extremes of atmospheric pressure changes can cause epistaxis.

Nasal dryness: This is caused either by dry air or a combination of dry air with septal deviations.



CT scan showing nasal spur

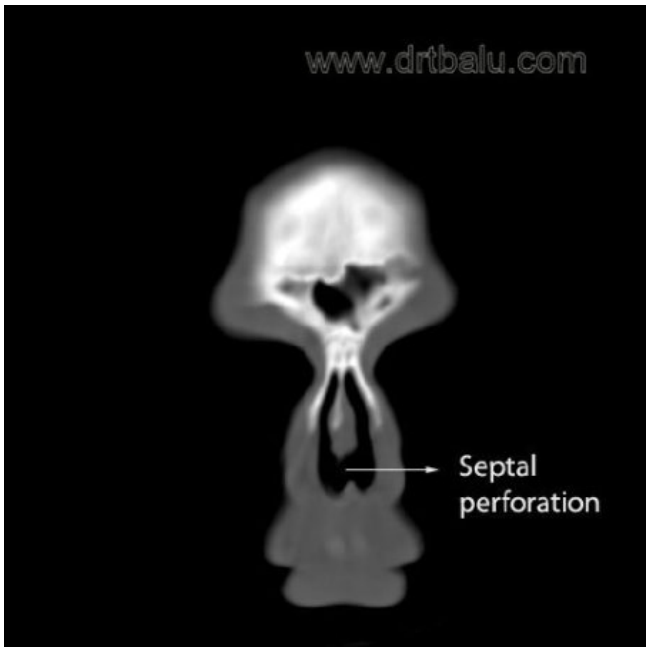
Fracture nasal bone being reduced Fracture nasal bone being reduced

Septal perforation:

This is one of the local cause of epistaxis. Perforated nasal septum causes excessive drying of edges of the perforation causing bleeding from the nose.

Septal perforation

CT scan showing septal perforation



Exposure to chemicals:

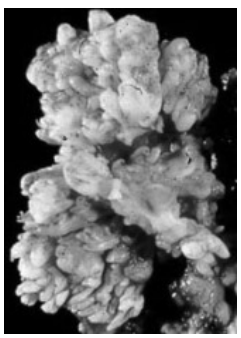
Cocaine abuse

Steroid spray

Decongestants (Rhinitis Medicamentosa)

Ammonia

Gasoline fumes, Chromium salts, Sulfuric acid

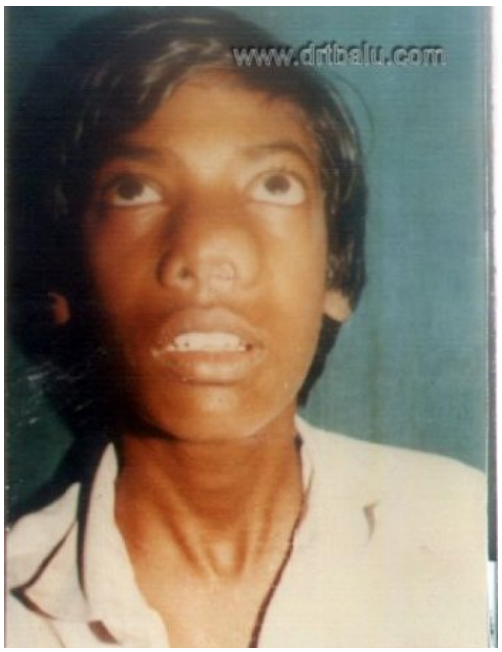


Inverted papilloma

Tumors:

Benign – Inverted papilloma, JNA, Septal angioma

Nasal mass



JNA mass

JNA mass seen in the nasopharynx

Malignant tumor maxilla

Malignant – Squamous cell carcinoma, esthesioneuroblastoma

Inflammation:

Presence of mucosal inflammation will cause epistaxis in these patients.

Atrophic rhinitis

Rhinosporidial mass

Rhinitis

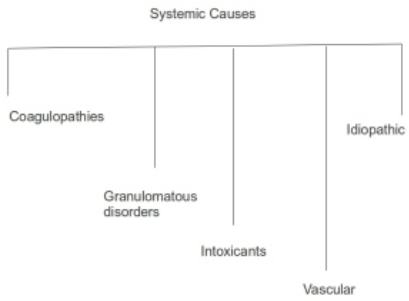
Sinusitis

Atrophic rhinitis [11]which is characterised by crusting of nasal mucosa, foul smelling nasal discharge, anosmia also causes epistaxis.

Rhinosporidiosis [12]caused by infection with Rhinosporidium seeberi presents with epistaxis.

Rhinolith

Systemic causes



Systemic causes:

Coagulopathies:

Anticoagulant / antiplatelet drug use

Haemophilia

Platelet defects

Pregnancy

Hepatic insufficiency

Alcohol ingestion

Scury

Haemorrhagic fevers

Granulomatous disorders – Wegener's disease, Midline granuloma, Syphilis, Tuberculosis, Rhinoscleroma, SLE, Periarteritis nodosa

Midline granuloma

CT scan nose showing rhinolith

Intoxicants:

Cobalt

Phosphorous

Arsenic

Lead

Vascular causes:

Hypertension

Atherosclerosis

Hereditary Haemorrhagic Telengiectasia

Management:

General assessment:

1. Assessment of airway

2. Vitals to be checked

3. Blood grouping and cross matching

Pinching the nose:

This is a useful first aid measure in patients with anterior episataxis. The patient is asked to pinch the nose while leaning forwards. Swimmer's nose clip [14]can also be used for this purpose. This provides constant localised pressure over the bleeding point and obviates the need to keep the nose pinched manually.

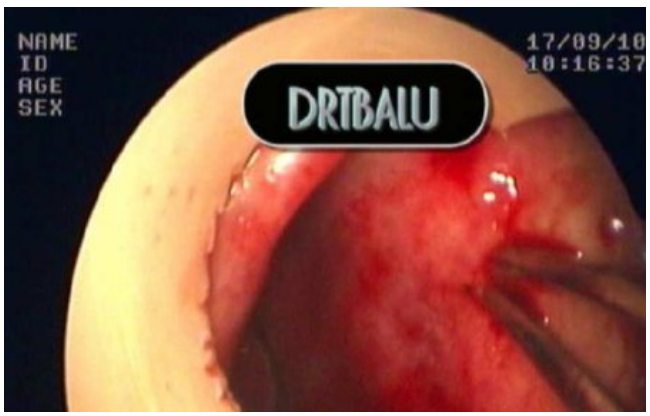
Nose clip

Management of anterior nasal bleed:

If the bleeding arises from Little's area / retrocolumellar vein area then cauterization may be resorted to. Bipolar cautery is ideal. In outpatient setup chemical cautery (Chromic acid, acetic acid) can also be tried out. Silver nitrate eventhough commonly used is not effective during active bleeds. Studies show that 30 seconds of exposure to chemical agent causes 1 mm penetration of tissue [13]. Cauterization should be precise. Random cauterization should be avoided at all costs as this could lead to troublesome septal perforation. After effective cauterization of the bleeding point patient is advised to use saline nasal spray to prevent excessive drying of nasal mucosa.

Use of nasal cream:

In children the most common cause of bleeding is from enlarged retrocolumellar vein. Constant nose picking could cause bleeding from this area. This area lies slightly anterior to the Little's area. Application of antiseptic cream [13]in this area would reduce the incidence of nasal picking in these children.



Bipolar cautery

Topical Haemostats:

Many topical haemostatic agents are available at present. These agents exert their effect by:

1. Improving primary haemostasis

2. Stimulating fibrin formation

3. Inhibiting fibrinolysis

4. Provides template for maintainance of endogenous coagulation

Types of Topical Haemostats:

1. Collagen based

2. Gelatin based

3. Cellulose based

4. Albumin derived

5. Inorganic haemostats

6. Fibrin based

7. Polymeric haemostats

Collagen based:

These haemostats were first introduced in 1970. These substances possess microfibrillar structure comprising of collagen molecules with non covalently bound hydrochloric acid. The molecular structure and the large surface area it provides are important for achieving haemostasis [15]. Contact with the bleeding area attracts platelets which gets entangled within the microfibrillar structure and degranulates there by promoting coagulation.

Gelatin based:

The mechanism of action of gelatin based topical haemostasis is not clearly understood. It promotes coagulation because of its surface effects. This may also be used alone or in combination with procoagulants. Floseal is a characteristic example. It contains gelatin based topical haemostatic and a procoagulant.

Cellulose based:

These have been in use for more than decades. Mechanism of action include:

1. Absorption of blood

2. Surface interactions with proteins and platelets

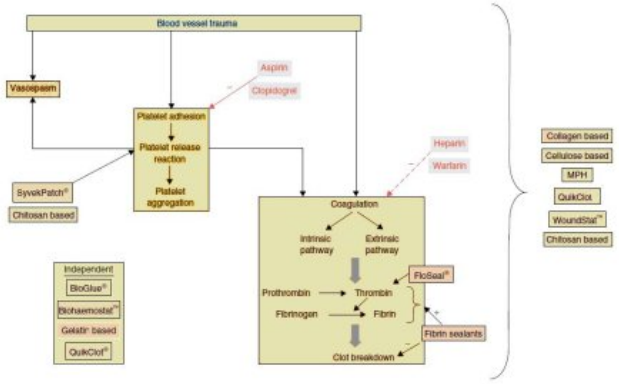
3. Activation of both intrinsic and extrinsic pathways[16]

Albumin derived:

Classic example of drug belonging to this category is gelatin–resorcinol–formaldehyde . It has been abandoned now due to toxic reactions caused by formaldehyde. Another haemostat of this category which is commonly used is Bioglue. In bioglue formaldehyde is replaced by less toxic gluteraldehyde. Advantage of this category of topical haemostats include its ability to cause coagulation outside both extrinsic and intrinsic pathways.

Ionic based:

This is actually a recent addition to the haemostat armamentorium. Most commonly used is Quickclot. This is based on zeolite. The mode of action is absorption of water from the bleeding site causing an increase in the concentration of platelets and coagulation factors.



Sites of action of local haemostats

Fibrin based:Fibrin based tissue adhesives has been in use since 1970′s. They have both hameostatic and adhesive properties and can be used to deliver antibiotics to the wound site. They have been reported to reduce adhesion formation and enhance wound healing.

Use of Gelfoam / Surgicel:

Absorbable haemostatic material like Gelfoam / surgicel can be used to pack the nasal cavity. This is very useful in managing patients with coagulopathy. Since this pack need not be removed mucosal trauma during removal can be avoided in these patients.

Gelfoam is actually a sterile haemostatic sponge prepared from purified Porcine skin gelatine. This is actually water insoluble. It has the capacity to absorb 45 times its weight of blood 17. Hence its absorptive capacity is directly proportional to its size [17]. The mechanism of haemostatic action of gelfoam is supportive and mechanical in nature. When applied to bleeding surfaces these substances arrest bleeding by forming artificial clots thereby providing the mechanical matrix thatfacilitates clotting mechanism 19. Clotting effects of gelfoam is due to release of thromboplastin by Platlets that come into contact with gelfoam 20. Gelfoam pack when applied to nasal cavity completely liquifies within 2-5 days.

Conventional nasal packing:

This is indicated in patients where cauterization is not possible / fails. Conventional nasal packing are of two types: Anterior nasal packing and post nasal packing.

Materials used for nasal packing:

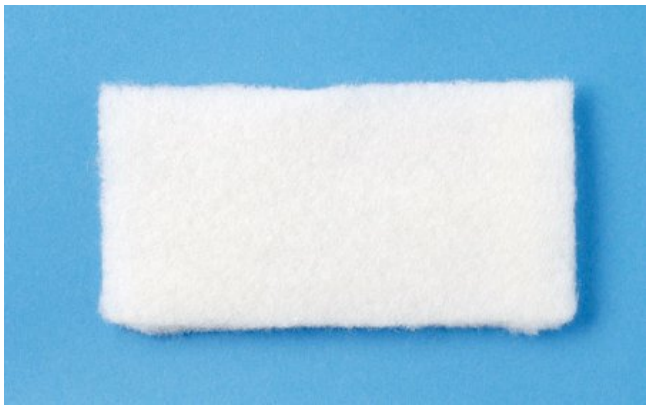
1. Roller gauze impregnated with antibiotic and lubricant like liquid paraffin

2. Merocel

3. Tampoons

4. Foley’s catheter for post nasal packing

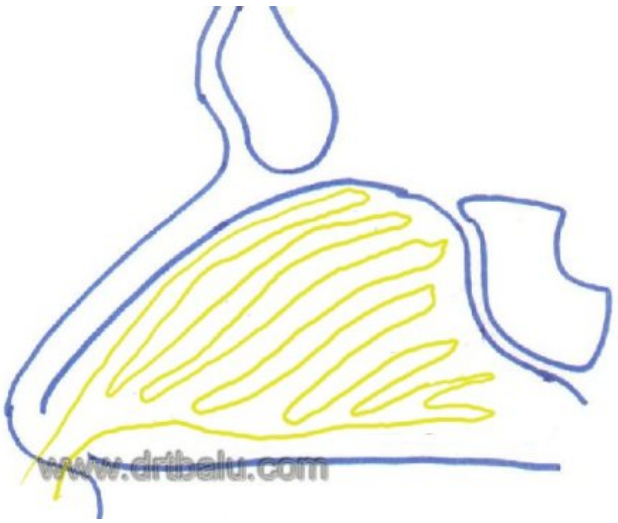
Merocel



If gauze is used for packing nasal cavity then it should be removed within 48 hours. If left in place for more days there is always an associated risk of nasal infection and toxic shock syndrome. If post nasal packing is resorted to then the patient should be admitted for close monitoring of oxygen saturation and airway observation.

Complications:

1. Headache
2. Obstruction to sinus drainage
3. Epiphora



Anterior nasal packing

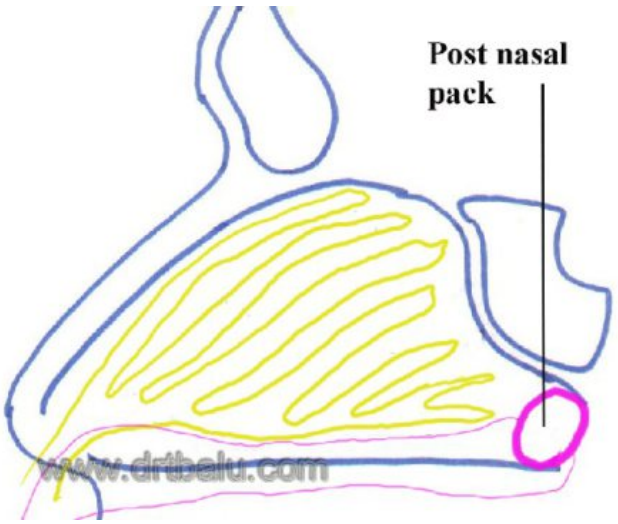
Merocel

Anterior nasal packing using roller gauze impregnated with liquid paraffin is sufficient to manage a majority of anterior nasal bleeds. The liquid paraffin acts as a lubricant, and as a moistoning agent. The tamponoding effect of a nasal pack is sufficient to stop nasal bleeding. This type of roller gauzes can be kept inside the nasal cavity only up to 48 hours after which it has to be removed and changed.

The newer packs like the BIPP (Bismuth Iodine paraffin paste) packs can be left safely in place for more than a week.

To manage post nasal bleed a post nasal pack is a must. Post nasal packing can be done in 2 ways:

Post nasal packing (conventional): A gauze roll about the size of the patient's naso pharynx is used here. Three silk threads must be tied to the gauze roll. One at each end and the other one at the middle. The patient should be in a recumbent position. After anesthetising the nasal cavity with 4% xylocaine the mouth is held open. Two nasal catheters are passed through the nasal cavities till they reach just below the soft palate. These lower ends of the catheters are grasped with forceps and pulled out through the mouth. The silk tied to the ends of the gauze is tied to the nasal catheters. The post nasal pack is introduced through the mouth and gradually pushed into the nasopharynx, at the same time the nasal catheters on both sides of the nose must be pulled out. When the pack snugly sits inside the nasopharynx, the two silk threads tied to its end would have reached the anaterior nares along with the free end of the nasal suction catheter.



Post nasal pack

The two silk threads tied to the suction catheters are untied. The catheters are removed from the nose. The silk thread is used to secure the pack in place by tying both the ends to the columella of the nose. The silk tied to the middle portion of the gauze pack is delivered out through the oral cavity and taped to the angle of the cheek. This middle portion silk will help in removal of the nasal pack. In addition to the postnasal pack anterior nasal packing must also be done in these patients.

Postnasal pack using baloon catheters: Specially designed baloon catheters are available. This can be used to perform the post nasal pack. Foleys catheter can be used to pack the post nasal space. Foley's catheter is introduced through the nose and slid up to the nasopharynx. The bulb of the catheter is inflated using air through the side portal of the catheter. Air is used to inflate the bulb because even if the bulb ruptures accidentally there is absolutely no danger of aspiration into the lungs. After the foleys catheter is inflated the free end is knotted and anchored at the level of the anterior nares.

Foley's catheter

Nasalballoons

Newer packing materials: Newer packing materials made of silicone are available. The advantages of these material are that they are not irritating, patient can breath through the nose with the pack on through the vent provided, these packs can be retained inside the nasal cavity for more than 2 weeks. They can be removed and repositioned ifnecessary The only disadvantage is that they are expensive.

Surgical management:

Endoscopic cauterisation can be tried if the bleeders are localised and accessible. If not accessible, ligation of the internal maxillary artery can be done through caldwelluc approach. Spenopalatine artery clipping can be done endoscopically. It is accessible close to the posterior end of the middle turbinate. In rare cases external carotid artery ligation at the neck can be resorted to. External carotid artery is differentiated from the internal carotid in the neck by the fact that internal carotid artery does not give rise to branches in the neck, while the external carotid artery does so.

Ethmoidal artery ligation: If epistaxis occur high in the nasal vault, anterior and posterior ethmoidal arteries may be ligated using ligaclips. These arteries can be accessed using an external ethmoidectomy incision. The anterior ethmoidal artery is usually found 22mm from the anterior lacrimal crest. If ligation of the anterior ethmoidal artery does not stop bleeding then posterior ethmoidal artery should also be ligated. The posterior ethmoidal artery can be found 12mm posterior to the anterior ethmoidal vessel.

TESPAL:

Tespal: (Trans nasal endoscopic sphenopalatine artery ligation)

History: This procedure was first reported by Budrovich and Saetti in 1992.

This procedure can safely be performed under GA. / L.A.

Indication:

Epistaxis not responding to conventional conservative management.

Posterior epistaxis

Procedure:

The nose should first be adequately decongested topically using 4% xylocaine mixed with 1 in 50,000 units adrenaline.

A 4mm 0 degree nasal endoscope is introduced into the nasal cavity. The posterior portion of the middle turbinate is

visualized. 2% xylocaine with 1 in 1lakh units adrenaline is injected in to this area to further reduce bleeding.

Incision: An incision ranging between 10 – 20 mm is made vertically about 5 mm anterior to the

attachment of the middle turbinate. The mucosal flap is gently retracted posteriorly till the crista ethmoidalis is visualized. The crista ethmoidalis is a reliable land mark for the sphenopalatine artery. The artery enters the nose just posterior

to the crista. The crista can infact be removed using a Kerrison's punch for better visualization of the artery.

The sphenopalatine artery is clipped using liga clip or cauterized as it enters the nasal cavity. This is done as close to the lateral nasal wall as possible, this would ensure that the posterior branches may also be reliable included.

Following successful ligation / cauterization, the area is explored posteriorly for 2 – 3 mm to ensure that no more

vessels remain uncauterized.

Nasal packing is not needed.

Complications of TESPAL:

1. Palatal numbness

2. Sinusitis

3. Decreased lacrimation

4. Septal perforation

5. Inferior turbinate necrosis

This procedure in combination with transnasal anterior ethmoidal artery ligation ensures that epistaxis is controlled reliably.

References

1. Cordes S. Epistaxis UTMB – Department of Otolaryngology Grand Rounds 1996

2. Ijaduola G.T.A., Okeowo P.A. Pattern of epistaxis in the tropics. Centr Afr J Med. 1983; 29 (4): 77-80

3. Michael S. Benninger , and Bradley F. Marple. Minor recurrent epistaxis; Prevalence and a new method for management. Journal of Otolaryngology - Head and Neck Surgery. Zwischenberger JB, Brunston RL Jr, Swann JR, Conti VR.

4. http://en.wikipedia.org/wiki/Ali_ibn_Sahl_Rabban_al-Tabari

5. http://www.drtbalu.co.in/epistaxis.html

6. Woodruff's plexus TW Chiu, J Shaw-Dunn etal The Journal of Laryngology & Otology/ Volume122 / Issue10 /October 2008, pp

7. Comparison of two topical collagen-based hemostatic sponges during cardiothoracic procedures. J Invest Surg 1999; 12: 101–106. 2004,131: 317-320

8. Beran, M, B. Petruson. Occurrence of epistaxis in habitual nose- bleeders and analysis of some etiological factors. ORL J Otorhinolaryngol Relat Spec. 1986;48:297-303.

9. Lennox, P. A., Harries, M., Lund, V. J., and Howard, D. J. A ret-rospective study of the role of the argon laser in the management of epistaxis secondary to hereditary haemorrhagic telangiectasia. J Laryngol Otol 1997; 111: 34-37.

10. Lund VJ, Howard DJ. A treatment algorithm for the managemenbt of epistaxis in hereditary hemorrhagic telangiectasia. Am J Rhinol. 1999; 13:319-322.

11. Hitchings AE, Lennox PA, Lund VJ, Howard DJ. The effect of treatment for epistaxis secondary to hereditary hemorrhagic telangiectasia. Am J Rhinol. 2005;19:75-78.

12. http://entscholar.wordpress.com/article/rhinosporidiosis/

13. Murthy P, Nilssen EL, Rao S, McClymont LG. A randomized clini-cal trial of antiseptic nasal carrier cream and silver nitrate cautery in the treatment of recurrent anterior epistaxis. Clin Otolaryngol Allied Sci. 1999;24:228-231.

14. Vaghela HM. Using a swimmer’s nose clip in the treatment of epistaxis in the A&E department. Accid Emerg Nurs. 2005;29.

15. Zwischenberger JB, Brunston RL Jr, Swann JR, Conti VR. Comparison of two topical collagen-based hemostatic sponges during cardiothoracic procedures. J Invest Surg 1999; 12: 101–106.

16. Kanko M, Liman T, Topcu S. A low-cost and simple method to stop intraoperative leakage-type bleeding: use of the vancomycin-oxidized regenerated cellulose (ORC) sandwich. J Invest Surg 2006; 19: 323–327

17. Council on Pharmacy and Chemistry: Absorbable Gelatin sponge — new and nonofficial remedies. JAMA 1947; 135:921

Evaluation of lacrimal apparatus

September 18, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Abstract

Currently otolaryngologists have started doing dacryocystorhinostomy using nasal endoscopes. This procedure done by an otolaryngologist has its own obvious advantages. The first and foremost being the need for external incision has been dispensed with. On the flip side otolaryngologist is not conversant with the examination techniques involving this area. Even the standard text books of otolaryngology are woefully inadequate in details regarding this subject. This e book discusses the examination techniques and investigations pertaining to rhinolacrimal system

Lacrimal apparatus – Its evaluation

Introduction:

Dacryocystorhinostomy as a treatment modality for epiphora is commonly being performed endonasally using a nasal endoscope by otolaryngologists. It is hence imperative that they diligently examine the entire lacrimal system before proceeding with the procedure. Despite the commonality of this surgical procedure, standard otolaryngology text books contain fewer literature on this subject. History of surgery for nasolacrimal pathway obstruction dates back to Hamurabi 2200 B.C [1]. Dacryocystorhinostomy is the undisputed treatment of choice for lacrimal system drainage obstruction below the level of common canaliculi. Endonasal approach of dacryocystorhinostomy was first described by Caldwell in 1893 [2].

Epiphora can be caused by blockage of lacrimal drainage system / excess lacrimation / loss of lacrimal pump mechanism. Lacrimal pump mechanism could be disrupted due to lower lid laxity or weakness of orbicularis oculi muscle. Normal lacrimation / or excess of it can be caused by irritation to cornea / conjunctiva [3]. This reflex is initiated by stimulation of trigeminal nerve.

Trigeminal stimualtion can be caused by:

1. Corneal foreign body
2. Keratitis
3. Conjunctivitis
4. Ocular surface disorders (dry eye)

Epiphora:

Greek terminology meaning “Downpour”.

This is defined as excessive watering of eye. This is invariably caused by obstruction to tear drainage. Causes of epiphora include:

Congenital: Congenital nasolacrimal duct obstruction. Incidence varies between 1-6% [4]. It is beleived that massage of entire naso lacrimal system releives obstruction in more than 90% [5]of cases. Majority of these obstruction resolve during the first year of life hence urgent surgical management is not necessary [6]. Probing is also known to be beneficial in these patients. The time of probing is controversial. Probing is advised up to the age of 5 in these patients [7].

Acquired:

1. Primary acquired nasolacrimal duct obstructions
2. Dacryocystolithiasis
3. Orbital / lacrimal trauma
4. Canalicular lacerations
5. Actinomyces within the canaliculi – Actinomyces are anaerobic gram positive bacilli resembling fungi. These organism are normal commensal of oropharynx. These organism are capable of causing cast – forming canaliculitis 9 leading onto lacrimal tract obstruction.
6. Canalicular infections following herpes infections / ectropion – Viral infections constitute a well recognised common cause of acquired canalicular obstruction [8]. These patients give history of an episode of blepharokeratoconjunctivitis before epiphora. Antivirals (idoxuridine) which are prescribed for this condition too add to the woes by causing more lacrimal obstruction. Naso lacrimal obstruction caused by antivirals are transient and disappear after the drug is discontinued. Herpes simplex viral infections are known to cause punctal changes.

Clinical examination goal in these patients is to distinguish between epiphora and lacrimation. While epiphora needs to be surgically managed medically. The focus should be in differentiating anatomical obstruction from functional disorders.

Anatomical obstruction:

Obstruction to the lacrimal drainage system is the feature to look for in this condition. Pathological changes could be seen involving the lacrimal sac, irregularities in lacrimal drainage system (canalicular stenosis, canalicular blockage, obstruction to nasolacrimal duct, diverticulous formation etc.) Lacrimal pathways can be obstructed due to internal derangements like inflammation of the epithelial linining. This is known as intrinsic obstruction. If lacrimal pathways are affected by

deforming lesions from outside like tumors causing compression to it has been termed as extrinsic obstruction.

Physiologic dysfuntion causing epiphora:

This is also known as functional epiphora. Here there are no anatomical changes to the lacrimal pathway. The functioning lacrimal pump mechanism is at fault. Pump mechanism can be affected in conditions like eyelid malpositions, eversion of lacrimal punctum, poor orbicularis oculi muscle tone as seen in patients with Bell's palsy.

Grading of epiphora:

The commonly used grading system was devised by Sahlin [9].

Grade	Degree of Epiphora
0	No epiphora
1	Epiphora only outdoors and during windy times
2	Outdoor epiphora No indoor epiphora
3	Outdoor and indoor epiphora

Fig. 1: Grades of epiphora

Anatomy of lacrimal system an overview [10]:

The lacrimal system consists of a superior and inferior puncta at the medial ends of upper and lower eyelids. These two drain into upper and lower canaliculi. These two canaliculi join to form the common canaliculus. This zone is known as the upper lacrimal system. The common canaliculus inturn leads into the lacrimal sac. The sac is about 12 – 15 mm long. It eventually narrows and leads into the nasolacrimal duct which drains into the inferior meatus of the nose. The naso lacrimal duct is about 18 mm long. The sac and the duct comprise the lower lacrimal system.

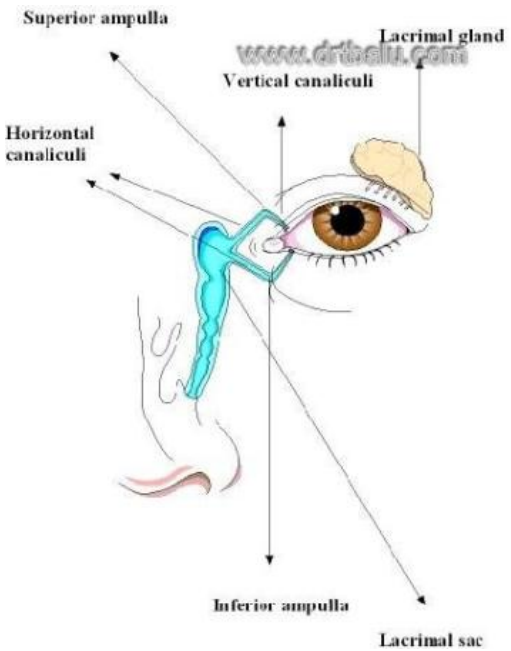


Fig. 2: Anatomy of lacrimal sytem

The junction between the common canaliculus and the lacrimal sac is guarded by the Rosenmuller valve. This valve prevents tear reflux. The nasal end of the nasolacrimal duct at the level of inferior

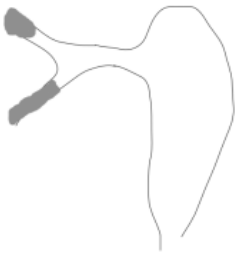
meatus is guarded by Hasner's valve.

Sites in the lacrimal system prone for obstruction:

Suprasaccal obstruction:

In this type obstruction lies proximal to the lacrimal sac. Obstruction can occur at the level of upper canaliculus, lower canaliculus and common canaliculus. Obstruction in these areas can occur following herpetic infections, taruma, irradiation.

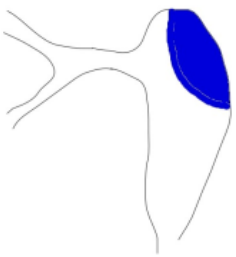
Suprasaccal obstruction



Saccal obstruction:

Here obstruction occurs at the level of lacrimal sac. This could be caused by tumors, diverticula, trauma etc.

Saccal obstruction

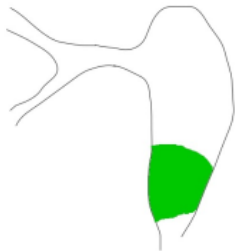


Subsaccal obstruction:

In this category the obstruction lies below the level of lacrimal sac. This condition commonly requires endoscopic dacryocystorhinostomy. This obstruction is more common than the rest.

Subsaccal incomplete obstruction

Complete subsaccal obstruction



Causes include:

1. Congenital nasolacrimal duct obstruction

2. Primary acquired nasolacrimal duct obstruction

3. Nasolacrimal duct obstruction following FESS

Functional obstruction:

This controversial term is used to explain those patients whose lacrimal system is patent to syringing but they still complain of epiphora. It should be borne in mind that the term obstruction

should be used only to indicate anatomical obstruction only.

Diagnosis of epiphora the Phylosophy behind it:

Theoretically speaking excessive tearing may be caused by -

Hypersecretion

Epiphora

Combination of both

Diagnostic evaluation should include:

1.Quantification of tear production

2.Assessment of nasolacrimal system patency

3.Differentiating epiphora from lacrimation

4.Defining the pathological process of epiphora

5.Differentiating anatomical from functional obstruction

6.Attempting to locate the obstruction in order to define the optimal surgical approach

Classification of Tests for lacrimal drainage pathway:

Anatomical tests

Functional tests

Secretory tests

Anatomical tests:

These tests are performed to locate the probable area of lacrimal tract obstruction. These tests include:

Palpation of lacrimal sac

Syringing / irrigation

Diagnostic probing

Dacryocystography

Nasal examination

CT / MRI

Functional tests:

These are performed to access the function of lacrimal apparatus under physiologic conditions.

This test is performed if there is no obstruction as evdenced by negative anatomical tests.

These tests include:

Flourescein dye disappearance test

Scintigraphy

Jones dye test I

Sacharin test

Tests of secretion:

These tests are performed to access secretory function of the lacrimal apparatus. These tests are performed in examining dry eyes. These tests include:

Schrimers test

Bengal Rose test

Tear-film break up

Tear lysozyme

Knowledge of various causes of lacrimation and epiphora really helps in clinical examination of these patients.

Excess lacrimation:

Supranuclear causes – Psychogenic / emotions

Stimulation of V cranial nerve – (Reflex tearing)

Lid causes (Blepharitis / Trichiaris)

Conjunctival diseases

Corneal diseases

Neuralgia

Ocular inflammation

Infranuclear causes – facial palsy, abberant innervation, crocodile tears

Lacrimal gland stimulation

Others – Bright lights, sneezing

Epiphora:

Functional insufficiency

Incorrect lid closure

Lid malposition

Punctal eversion

Punctal medialization

Anatomical obstruction

Combined lacrimation / epiphora – A combination of the above two categories

Facial nerve palsy – Corneal irritation and pump defects

Lower lid ectropion – Conjunctival irritation , ineffective pump mechanism

Thyroid diseases – Corneal irritation, defective canalicular function

Clinical history:

This is a very important aspect of lacrimal apparatus examination. This will provide vital clues to

the presence of canalicular disorders 12. History should include patient's present and past ophthalmological problems, nasal symptoms, medical and interventional relevant procedures also.

Unilateral tearing usually indicate obstructive pathology whereas bilateral tearing could be physiological. A child with a history of tearing since birth should arouse suspicion of membranous obstruction to nasolacrimal duct. Nasal disorders like nasal polyposis / sinusitis can also cause

unilateral epiphora.

Inspection and palpation should involve the following areas:

1. Eyelids

2. Medial canthus

3. Palpation of lacrimal sac

Eye lid examination:

Look out for lower eyelid laxity

Ectropion

Punctal eversion

Trichiasis

Blepharitis

Snap-back test – This test is performed by pulling the lower eye lid down and away from the globe and held for several seconds. On release the lower lid resumes its normal position. The time taken for resumption of normal position is noted. The patient should not blink during the test. This test provides an assessment of laxity of lower lid. The longer it takes for the lower lid to spring back to

position the more lax it is. This test is graded on a scale of 4 starting from 0. 0= normal and 4= lax lower lid.

Medial canthal laxity

Lateral canthal laxity

Orbicularis oculi muscle tone check

Pinch test – This test helps to assess orbicularis oculi muscle tension.

Examination of medial canthus:

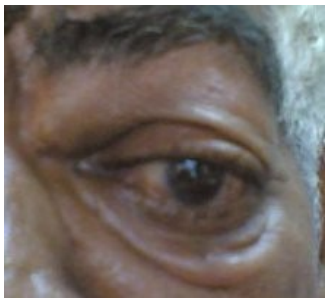
Lacrimal sac enlargement will be seen as mass below medial canthal tendon.

Enlargement above medial canthal tendon indicates neoplasm.

Palpation of lacrimal sac:

Normal sac is not palpable. Sac swelling is usually confined to below the medial canthal tendon. If there is neoplasm then it is likely to extend above the medial canthus.

Reflux of tears / pent up mucopurulent secretions can be seen on palpating the lacrimal sac area.



Swelling above medial canthus

Pain and tenderness over this area indicates acute dacryocystitis.

Sac being squeezed off its secretions

Sac swelling

Dye excretion tests:

These tests help in ascertaining drainage functions and patency of the entire nasolacrimal system.

Fluorescein dye is used for this purpose. This test is considered to be more physiological [11]since the lacrimal system is not instrumented and the dye flows along with tears through the normal passages. The principle of this test is evaluation of residual fluroescein dye in the eye following instillation of one drop of it into the unanaesthetized conjunctiva.

Caution: This test doesnot distinguish anatomical from functioning defects [12].

In performing this test one drop of 1% fluorescein is instilled into the lower fornix of each conjunctival sac. After 5 mins, the thickness of fluorescein of the tear meniscus is measured using cobalt blue filter. Studies reveal that it takes 5 mins for tears to normally drain through the system.

This test can safely be performed in infants and children [13].

Presence of fluorescein gives no information on the localisation of obstruction. Presence of residual

fluorescein is an indication for probing and syringing. When performing this test in infants the child should be held in a vertical position.

Dye test grading:

0=No fluorescein in the conjunctival sac

1=Thin flurescing marginal tear drop persists

2=More fluorescein persists somewhere between 1 and 3 grades

3=Wide brightly fluorescein tear strip

Among these grades 0 and 1 are considered normal

False negatives can occur in:

1. Large lacrimal sac

2. Mucocele

3. Distal nasolacrimal duct block

Break up time test:

This test is performed by placing a drop of fluorescein in the external canthus in the lower eyelid.

Its transport can be observed from lateral to medial across the eyelid and into each punctum. Holes in the tear film can be observed. This is break up time test.

Jones dye tests: This group of tests are used to distinguish between functional and anatomic outflow problems. The primary test is performed by placing topical anesthetic and flurescein dye into the conjunctival sac. Topical 4% xylocaine and oxymetazoline nasal sprays may be used to anesthetize and vasoconstrict the inferior meatus of the nose. A cotton tipped applicator is placed beneath the

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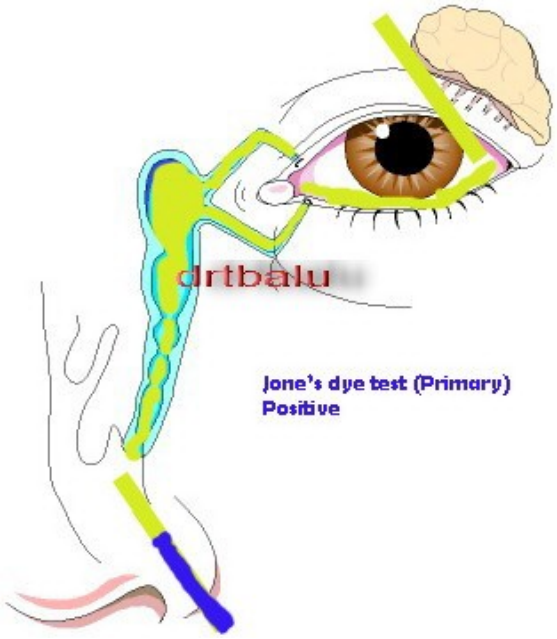
Jones dye test



inferior turbinate near the opening of the nasolacrimal duct. Recovery of fluorescein dye in the nose indicates a functionally and antomically patent system. Non recovery of the dye (negative result) suggests a functional or anatomic blockage.

In the event of negative dye test, secondary dye test should be performed. This test is performed after removal of residual fluorescein from the conjunctival sac. Clear saline solution is placed into the inferior canaliculus using a syringe / cannula. The irrigant is retreived from the nasal cavity by tilting the patient's head forward over a basin. If fluoresein dye is present in the irrigant (positive

result) then it is assumed that the upper lacrimal system is functional while the lower system is partially open and is not functional. Recovery of a clear irrigant (negative result) indicates a functional problem with the upper system.

Caution: This test is useless in patients with total lacrimal tract obstruction. This test should be performed only if the lacrimal system is patent for syringing.

Saccharin test:

This test is more or less similar to fluorescein dye test. This test is also hence physiological. A drop of saccharin is placed into anesthetized and the time taken for the patient to taste saccharin is measured. Approximate time is about 3.5 mins. The flip side in this test is that the patient should

have normal taste sensation.

Diagnostic probing and lacrimal syringing:

These are invasive tests. They provide valuable information on location of obstruction. They establish diagnosis of anatomical obstruction in the lacrimal system. This test is virtually useless in functional obstruction [14]. Syringing / irrigation of lacrimal system is not a physiological test since the pressures used is more than the normal pressure of lacrimal syste. Hence this test should be

interpreted with fluorescein dye test and clinical examination.

Procedure:

1. Topical 4% xylocaine drops applied to the conjunctiva

2. Punctum dilator is used to dilate the punctum and ampulla

3. A blunt cannula is placed in the inferior canaliculus. The lower eyelid is pulled down to straighten the inferior canaliculus. Superior canaliculus is gently stretched laterally prior to irrigation

4. Tip of the irrigator is placed in the inferior canaliculus, first vertically and then horizontally

with the eyelid on stretch. The tip is advanced 3-7 mm into the canaliculus and sterile saline

is injected.

5. It is important to avoid forced irrigation to avoid damage to the canaliculi

Interpretation: Regurgitation of irrigated saline through the opposite punctum indicates an obstruction in the common canaliculus or more distal structures. Regurgitation of fluid via the same canaliculus indicates punctal obstruction and syringing should be repeated via the opposite canaliculus. Irrigation into the nose indicates normal drainage function. It does not rule out functional obstruction.

Probing (Diagnostic):

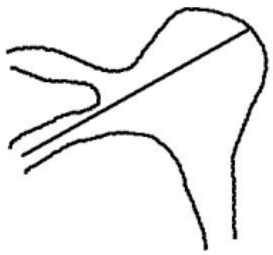
This test should be performed if syringing test indicate obstruction and the location of the obstruction is to be ascertained. Obstruction can be located in the canaliculi and their assessment is vital in deciding the mangement modality in these patients. If irrigated fluid regurgitates through

opposite punctum obstruction of common canaliculus or more distally is possible. The exact site in this scenario could be ascertained by careful probing of the entire system. Probing can be performed using blunt Bowman's probe which come in various sizes.

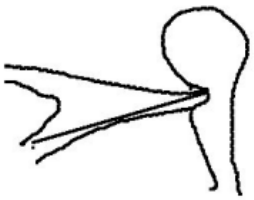
Procedure:

After instilling topical anesthetic drops into the conjunctiva the punctum is dilated using lacrimal

Fig. 3: Hard stop



Soft stop



probe. The probe is then passed vertically and then horizontally with the eyelid in stretch till the lacrimal bone is encountered or soft obstruction is reached. If the probe encounters lacrimal bone then it is known as hard stop. This is actually normal. If the probe encounters obstruction then it is known as soft stop. If irrigation showed reflux through the opposite punctum and the probe encounters hard stop then obstruction could be at the level of lacrimal sac or nasolacrimal duct.

Radiological evaluation:

Include:

Dacryocystography

Nuclear lacrimal scintigraphy

CT

MRI

Dacryocystography:

This is an anatomical investigation. This is indicated when there is block in the lacrimal system as indicated by syringing test. It helps in creating an internal image of the entire lacrimal system. In this test radio opaque water soluble dye is injected either into upper / lower canaliculus and magnified images are taken. Using digital subtraction techniques excellent images of the entire lacrimal system can be ensured.

Radiologic criteria of lacrimal pathology [15]:

1.Regurgitation of radio-opaque fluid into the conjunctival sac

2.Absence of fluid in the nose

3.Fluctuation of lumen of lacrimal system

4.Irregularity in contrast

5.Deformation involving lacrimal sac

Interpretation of irrigation and probing results

	Liquid in nose/pharynx	Reflux into lower canaliculus	Reflux into upper canaliculus	Result
Hard stop	yes	No	No	Normal
	No	No	Yes	Nasolacrimal duct stenosis
	No	No	No	Subsaccal obstruction
Soft stop	No	Yes	No	Medial canalicular/common canalicular block
	No	No	Yes	Distal common canalicular block

Nuclear lacrimal scintigraphy:

This is a non invasive physiological test. This test utilizes radiotracer technitium-99M pertechnitate. This can be analysed using a gama camera. This is useful only in patients whose lacrimal system is patent on syringing despite epiphora. This is found to be useful in difficult cases

and incomplete obstruction.

This test is performed without instilling topical anesthesia. A drop of technetium-99m is instilled into each conjunctival sac of a patient sitting in front of a gamma camera. Normal blinking of eyes are allowed. Patient stares at a distant target during a 20 mins test period while images are being

recorded with a gamma camera.

CT scan/ MRI scan:

Helpful in identifying tumors involving sac, or adjacent areas.

Rhinosporidiosis of lacrimal sac

Secretory tests:

These patients are useful in evaluating those with complaints of dry eye.

Schirmer's test: This test is basically prepared to quantitate tear production. This test is performed by placing strips of white filter paper 35×5 mm at the junction of the middle and lateral thirds of the lower eyelids after administration of a topical anesthetic agent. The tear production is measured

Schrimer's test

with the eyes closed. Produced tears will wet the filter paper. The length of the filter paper which becomes wet is assessed at the end of 5 minutes. Normal test result is between 10mm and 30 mm of wet filter paper. Normally it should not exceed 30 mm. A value of more than 30 mm is considered to be epiphora. A value of less than 10 mm is considered to be dry eye (hyposecretion).

Breakup time test:

This test indicates function of mucin layer / reflex hypersecretion of aquous component of the tears.

One drop of fluorescein is instilled into the external canthus of a lower lid and the patient is instructed to blink once and then to keep his eyes open. The holes developed in the tear film are observed at the cornea through a slit-lamp with illumination through the cobalt filter. The normal

breakup time should be approximately 15–30 s. A break-up time of less than 10 s indicates a deficiency and the epiphora should be treated with libricating eye drops .

Bengal Rose test:

This test is also similar to that of Break up time test. One drop of Bengal Rose dye is placed in the conjunctiva and the patient is instructed to blink several times within a minute. Interpalpebral

staining is seen in patients with dry eye.

Lysozyme lysis test:

The amount of a lysozyme activity and concentration is decreased in hypersecretion and in hyposecretion, and it usually precedes clinical symptoms. A lysozyme activity (and concentration)

is estimated on the basis of the inhibition of the growth of the bacterium Micrococcus lysodicticus.

References

1. Onerci M. Dacryocystorhinostomy. Diagnosis and treatment of nasolacrimal canal obstructions. Rhinology 2002; 40:49-65.

2. Cokkeser Y, Er H. Comparative external vesus endoscópico dacryocystorhinostomy. Otolaryngol

Head and Neck Surg 2000; 123:488-91

3. Hurwitz JJ (1996) The lacrimal system. Lippincott-Raven Publishers, Philadelphia

4. Guerry D, Kendig EL. Congenital impotency of the nasolacrimal duct. Arch Ophthalmol 1948, 39: 193-204.

5. Price HW. Dacryostenosis. J Pediatr 1947, 30: 302-305

6. Ballard EA.Excessive tearing in infancy and early childhood: the role and treatment of congenital nasolacrimal duct obstruction. Postgrad Med 2000; 107:149-54

7. Kashkouli MB, Kassaee A, Tabatabaee Z. Initial Nasolacrimal Duct Probing in Children under Age 5: Cure Rate and Factors Affecting Success. J AAPOS 2002; 6:360-3

8. Bouzas, A. (1965). Canalicular inflammation in ophthalmic cases of herpes zoster and herpes simplex. American Journal of Ophthalmology, 60, 713-716.

9. Sahlin S, Rose GE. (2001) Lacrimal drainage capacity and symptomatic improvement after dacryocystorhinostomy in adults presenting with patent lacrimal drainage systems. Orbit, 20:173-179.

10. http://www.drtbalu.com/Endo_dcr.html

11. Meyer DR, Antonello A, Linberg JV (1990) Assessment of tear drainage after canalicular obstruction using fluorescein dye disappearance. Ophthalmology 97:370–374

12. Hurwitz JJ (1996) The lacrimal system. Lippincott-Raven Publishers, Philadelphia

13. Lloyd GAS, Welham RAN (1974) Subtraction macrodacryocystography. Br J Radiol 47:379–382

14. Sahlin S, Chen E (1996) Evaluation of the lacrimal drainage function by the drop test. Am J Ophthalmol 122:701–708

15. Walther EK, Köster O, Straehler-Pohl HJ (1989) Dakryozystographie in digilater Subtraktionstechnik. Laryngol Rhinol Otol 68:396–400

Intrinsic Rhinitis

December 20, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Abstract

Intrinsic rhinitis is defined as a non infective and non allergic condition characterised by nasal block, rhinorrhoea and hyposmia. This is purely a medical condition. Awareness of this condition will help us to avoid unnecessary surgical procedures on patients suffering from this disorder. Surgery should be reserved only for cases that are intractable to medical management. This article discusses the complete gamut of this disorder.

Intrinsic Rhinitis

Introduction:

Rhinitis is inflammation of nasal mucosa characterized by nasal discharge, itching and congestion. It affects 20% of the population[1] .

Intrinsic rhinitis is defined as a non infective and non allergic condition characterized by nasal block, rhinorrhoea and hyposmia. This is purely a medical condition.

Intrinsic rhinitis encompasses two separate disease entities [2]. These entities show:

1. inferior turbinate hypertrophy

2. nasal polyp formation.

symptoms

Symptoms of intrinsic rhinitis

Symptom	Eosinophilic	Non eosinophilic
Obstruction	Moderate / severe	Mild
Rhinorrhoea	Mild / Moderate	Severe
Sneezing / Pruritis	Minimal	Minimal
Hyposmia	Usual	Rare
Mucosal swelling	Marked	Mild
Inferior turbinate enlargement	Marked	Mild
Polypi	common	Never

Sinus mucosal thickening	Common	Rare

Clinical presentation:

Rhinitis is generally characterised by 6 main symptoms: They are

1. Congestion

2. Sneezing

3. nasal itching

4. rhinorrhoea

5. hyposmia

6. post nasal discharge

Among these main symptoms nasal itching and sneezing are features of allergic rhinitis and hence are not seen in intrinsic rhinitis. All the other symptoms are manifested in intrinsic rhinitis.

Seebohm identified two groups of patients amongst those suffering from perenial rhinitis. One group had eosinophils in their nasal secretions while the other did not have any eosinophils in their nasal secretions. Accordingly he classified intrinsic / perenial rhinitis into eosinophilic and non eosinophilic types.

Eosinophilic group: This group is characterised by marked nasal congestion, profuse rhinorrhoea, hyposmia, inferior turbinate hypertrophy and mucoid nasal secretion. Nasal polyposis frequently occurred in this group of patients.

Non eosinophilic group: In these patients nasal obstruction is very mild, rhinorrhoea is very severe. They do not have significant mucosal swelling. Inferior turbinate hypertrophy is not significant. Tendency of nasal polyp formation is rare in this group.

Aetiology of intrinsic rhinitis:

Theories regarding aetiology of intrinsic rhinitis are:

1. Autonomic imbalance

2. Airway hyperreactivity

3. Allergic reaction to unidentified allergen

4. Disturbances of Beta receptor function

Mechanisms of Beta receptor dysfunction:

1. Down regulation caused by excess endogenous noradrenaline stimualtion.

2. Down regulation and uncoupling of adenylate cyclase producedby the inflammatory mediator induced activation of protein kinase.

3. The action of Beta receptor inhibitory factor presumed to be an anti beta receptor autoantibody.

4. Dysfunction of Beta receptor kinase causing short term desensitisation of beta receptors after

exposure to beta agonists.

Role of autonomic nervous system in causing intrinsic rhinitis:

The autonomic nervous system exerts its effects by secreting neurotransmitters ar their nerve endings. The neurotransmitters secreted are adrenaline, noradrenaline, vasoactive intestinal polypeptide, acetylcholine and neuropeptide Y.

The nasal resistance to air flow is controlled by sympathetic system, whereas the nasal glands are innervated by parasympathetic nerves. Increased parasympathetic outflow causes glandular hypersecretion. Vaso active intestinal polypeptide has been known to cause this effect.The vasodilatation caused due to the effects of vaso active intestinal polypeptide is resistant to the effects of atropine.

Management:

Majority of patients with intrinsic rhinitis benefit from medical management. Only a few require

Medical management of intrinsic rhinitis:

Topical iso tonic saline spray can be used for both forms of intrinsic rhinitis. Saline spray causes a reduction of post nasal drip, sneezing and nasal congestion [3].

Topical intranasal administration of Capsaicin (derived from pepper). This irritant chemical desensitizes the sensory nerve endings of the nasal mucosa thereby reducing nasal hyperactivity [4].

Eosinophilic type:

Steroids – Topical e.g. fluticasone, budesonide. A short course of systemic steriods can be administered.

Alpha receptor agonists – Systemic e.g. pseudoephidrine Topical e.g. xylometazoline (short course)

Mast cell stabilisers – Topical cromoglycate solution.

Non eosinophlic type :

Anti cholinergic – Topical e.g. ipratropium Hyosine administered orally or as a patch.

Anti cholinergic / sympathomimetic – Imipramine orally, chlorpheniramine orally.

Surgical Management

Surgical management of Intrinsic rhinitis

Symptom	Aim	Surgery
Nasal obstruction Rhinorrohea	Turbinate reduction Turbinate resectionVidian neurectomy	Submucosal diathermy CryosurgeryLaser surgeryPartial resectionSubmucosal turbinectomyRadical turbinectomyExcision of vidian nerveEndoscopic vidian neurectomy

References

1. Powe DG, Huskinsson RS, Carney AS, et al. Evidence for an inflammatory pathophysiology in idiopathic rhinitis. Clin Exp Allergy 2001;31:864 - 9.

2. Settipane RA, Settipane GA. Nonallergic rhinitis. In: Kaliner MA, ed. Current Review of Allergic Diseases. Philadelphia: Current Medicine, 1999.

3. Bronsky EA, Druce H, Findlay SR, Hampel FC. A clinical trial of ipratropium bromide nasal spray in patients with perennial nonallergic rhinitis. J Allergy Clin Immunol 1995;95:1117-1122.

4. Stjärne P, Lundblad L, Änggard A, et al. Local capsaicin treatment of the nasal mucosa reduces symptoms in patients with nonallergic nasal hyperreactivity. Am J Rhinology 1991;5:145-151.

Local anaesthesia of Nose and nasal cavity

September 23, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Introduction:

Anaesthesia of nose and nasal cavity are indicated for various diagnostic and surgical procedures involving the nose. Some of these indications include:

1. Insertion of Ryles tube

2. Diagnostic nasal endoscopy

3. Repair of fracture nasal bone[1]

4. Nasal packing for epistaxis

5. Foreign body removal

6. Abscess drainage / Septal hematoma drainage

7. Nasotracheal intubation[12]

Types of anaesthesia:

1. Topical anaesthesia using 4% topical xylocaine / 10% typical xylocaine spray[3]

2. Infiltration anaesthesia using 2% xylocaine

3. Regional blocks

4. Combination of these

Innervation of nose:

For effective admininstration of local anaesthesia a complete understanding of sensory innervation of nose and nasal cavity is a must. Innervation of nose can be divided into:

1. Innervation of mucosa within the nasal cavity

2. Innervation of external nose and its skin covering

Sensory innervation of external nose:

External nose and its skin lining is innervated by ophthalmic and maxillary divisions of trigeminal nerve.

Superior aspect of the nose is supplied by – Supratrochlear and Infratrochlear nerves (branches of trigeminal nerve) and external nasal branch of anterior ethmoidal nerve.

Inferior and lateral parts of the nose – is supplied by infraorbital nerve.

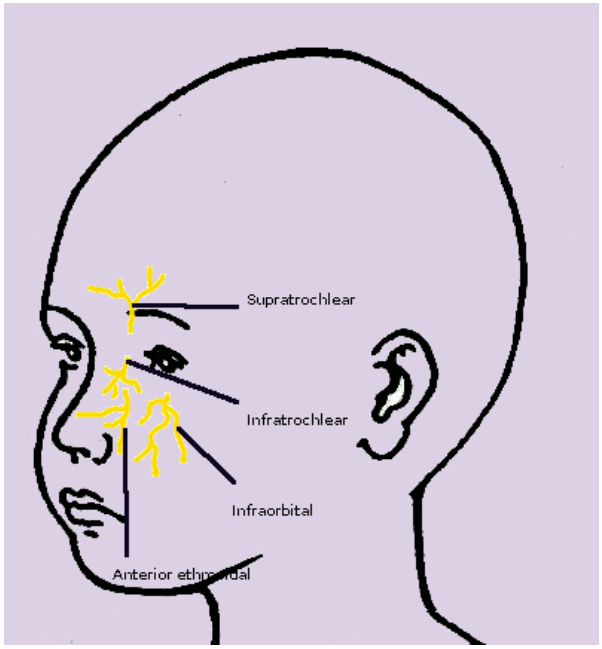


Fig. 1: Sensory Innervation of External Nose

Sensory innervation of interior of nasal cavity:

1. Superior inner aspect of the lateral nasal wall is supplied by anterior and posterior ethmoid nerves

2. Sphenopalatine ganglion present at the posterior end of middle turbinate innervates the posterior nasal cavity

3. Nasal septum is supplied by anterior and posterior ethmoidal nerves. Sphenopalatine ganglion also contributes to the sensory supply to the nasal septum via its nasopalatine branch.

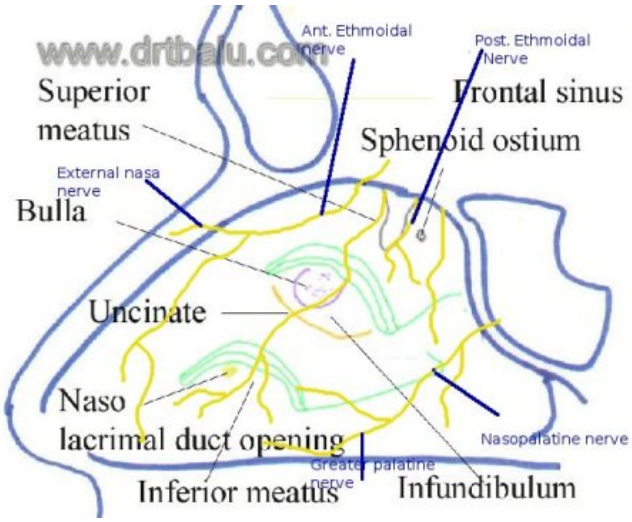
4. Cribriform plate superiorly holds the olfactory special sensation fibers.

Mucosal surface anaesthesia can be achieved by:

1. Using 10% xylocaine nasal spray – Topical surface anaesthesia just lasts for about 45 mins. This type of anaesthesia is preferred while performing diagnostic nasal endoscopy / minor procedures involving the nasal cavity like nasal packing.

2. Nasal packing using cottonoids / pledgets soaked in 4% xylocaine mixed with 1 in 10000 adrenaline is useful for performing minor surgical procedures inside the nasal cavity. Cottonoids are comparatively better than cotton pledgets. Each nasal cavity should be packed with 3 packs. One is placed in the floor of the nasal cavity, the next one is placed over it to encroach into the middle meatus and the last one is placed above the second one to anaesthetize the frontal recess area. Presence of adrenaline in the mixture shrinks the nasal mucosa and prolonges the duration of topical anaesthesia.

3. Infiltration anaesthesia is preferred while performing surgeries inside the nasal cavity. 2% xylocaine mixed with 1 in 10000 adrenaline is used for infiltration. Infiltration can be used to anaesthetize the anterior ethmoidal nerve, infraorbital nerve via the canine fossa. This is very useful during reduction of fracture nasal bone.



Sensory innervation of nasal mucosa

Caution:

While using 4% topical xylocaine for anaesthesia the maximum volume of the drug used should not exceed 7 ml. Posterior pharyngeal wall mucosa gets anaesthetized when pledgets dipped in 4% xylocaine is used to pack the nose. This may cause the patient to aspirate blood and secretions.

Periodical suction should be applied to the patient's throat while performing nasal surgeries under local anaesthesia.

References

1. Waldron J, Mitchell DB, Ford G. Reduction of fractured nasal bones; local versus general anaesthesia.Clin Otolaryngol Allied Sci. Aug 1989;14(4):357-9

2. Techanivate A, Leelanukrom R, Prapongsena P, Terachinda D. Effectiveness of mouthpiece nebulization and nasal swab stick packing for topical anesthesia in awake fiberoptic nasotracheal intubation. J Med Assoc Thai. Oct 2007;90(10):206371

3. Stolz, D., et al., Nebulized lidocaine for flexible bronchoscopy: a randomized, double-blind, placebo-controlled trial. Chest, 2005. 128(3): p. 1756-60.

Reducing bleeding during FESS

September 10, 2012 · Rhinology

Authors

Balasubramanian Thiagarajan

Abstract

This article discusses the importance of anesthesia in endoscopic sinus surgery. Major aim of anesthetist in FESS should be to reduce blood pressure to such a level that bleeding is minimized. It also discusses the various steps that should be followed by surgeons to reduced intra operative bleeding during the procedure.

Introduction:

Bleeding is one complication that could increase the risk of complication during endoscopic sinus surgery.

Considerable amount of attention should be paid to reduce bleeding on the table during the surgical

procedure. Dry surgical field not only improves visibility during endoscopic sinus surgery, it also shortens the

duration of surgery. In this regard both anesthesiologist and the operating surgeon have a vital role to play.

Endoscope becomes rather useless when the operating field bleeds. Bleeding is more common if surgery is

performed on allergic / inflamed nasal mucosa. This is where operating surgeon should take extra precaution

in preparing the patient. Reduction of nasal allergy and inflammation is also known as mucosal preparation

prior to surgery. This is done by administering a course of antibiotic, antihistamine and topical steroid spray [1].

Ideally patient should be prescribed these medications at least 1 week prior to surgery.

Bleeding is more common close to large vessels. Stamberger[2] has included 4 areas which are responsible for

extensive bleeding during endoscopic sinus surgery.

1. Anterior ethmoidal artery located in an osseous channel close to ethmoid roof

2. Branch of sphenopalatine artery close to the posterior end of middle turbinate. This is more prone for

injury in patients with well pneumatized middle turbinate (concha bullosa)

3. Damage to sphenopalatine artery while attempting to widen the sphenoidal ostium

Classification of surgical bleeding during endoscopic sinus surgery:

Surgical bleeding during FESS has been classified into:

Arterial

Venous

Capillary

Out of these three types of bleeding it is the capillary bleed that causes the most trouble during FESS[3].

Capillary bleeding can be reduced considerably by careful packing of the nasal cavity with cotton pledgets /

neuropatties soaked in 4% xylocaine mixed with 1 in 10000 adrenaline. This concentration is being used by

the author with great degree of success. The concentration of adrenaline is the source of raging controversy.

One aspect should be clearly borne in mind, never exceed 7 ml of 4% xylocaine while packing. Any volume

about 7ml should prove to be toxic to the patient.

Position of the table:

This plays a vital role in reducing capillary bleed. If the surgical field is kept above the level of the heart blood

flow to the heart is considerably reduced. This is also known as postural ischemia. Systolic pressure has

been estimated to reduce by 2 mm of mercury for every 2.5 cms of head elevation[4]. Ideally during

endoscopic sinus surgery is the head of the patient is in an elevated position capillary bleeding will be reduced

a lot.

Maintaining normal body temperature[5]:

During surgical procedure maintaining normal body temperature is very important. Significant levels of hypo /

hyperthermia can affect platelet function causing increased bleed during the procedure.

Role of anesthesiologist:

Anesthesiologist play a vital role in reducing blood loss during surgery. Bleeding is directly proportional to the

mean arterial pressure. As long as the mean arterial pressure is held within a low range bleeding will be

minimal. Use of intravenous anesthetic agents like Propofol can reduce bleeding when compared to

that of

conventional inhalational agents. Propofol is known to reduce brain metabolism and its circulation. Maximum

bleeding during Fess occurs from central vessels. Thus it plays a vital role in reducing bleeding. Use of

propofol with fentanyl supplementation actually serves the purpose. Even sevoflurane6 is known to increase

bleeding during Fess.

Use of Nitroglycerine infusion:

Nitroglycerine infusion during surgery causes prolonged hypotension. Only flip side to the use of NTG drip is

compensatory tachycardia which can push up the blood pressure. This tendency of compensatory

tachycardia is commonly seen in young individuals. This can be obviated by putting the patient on

preoperative night dose of beta blockers. Captopril can be used on the table to reduce the hear rate that

could occur due to NTG drip.

If the patient is under prolonged hypotension, their status should be meticulously monitored.

References

1. Preoperative treatment with topical corticoids and bleeding during primary endoscopic sinus surgery. Albu S, Gocea A, Mitre I.Otolaryngol Head Neck Surg. 2010 Oct;143(4):5738.

2. Stammberger H. Functional endoscopic sinus surgery: the messerklinger technique. Philadelphia: BC Decker; 1991

3. Jacobi KE, Bohm BE, Rickauer AJ, Jacobi C, Hemmerling TM. Moderate controlled hypotension with sodium nitroprusside does not improve surgical conditions or decrease blood loss in endoscopic sinus surgery. Clin Anesth

4. Enderby GE. Pharmacological blockade. Postgrad Med J 1974;50:5725

5. Schmied H, Kurz A. Mild hypothermia increase blood loss and transfusion requirements during total hip arthroplasty. Lancet 1995;347(8997):2892.